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SUDDEN DEATH OR ALLEGED ACCIDENTAL SUFFOCATION IN BABIES.

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DEATH in the form of natural disease sometimes strikes with great rapidity in young babies, leaving little to find at autopsy, and the death may be miscalled "accidental suffocation". A highly important question at once confronts us: Do babies accidentally suffocate in the bed clothes or face downwards on the bedding? The object of this paper is to examine this question briefly and to draw attention to the fact that so-called accidental suffocation by the bed clothes may be in reality swift death from undiscovered natural causes. We believe that it is.

We are not concerned here with criminal action and suspicious circumstances, and exclude accidents of the type in which the infant is strangled in his bed by restraining harness, or has his head jammed between the bars or beneath the drop side of his cot, or as occasionally happens, has his head wedged between a badly fitting mattress and cot margin, accidents of rare occurrence.

In the area which comes within the jurisdiction of the Melbourne coroner, on the average about thirty babies are found dead in their cots each year in private homes. These babies are thought by the parents to be well (it is usually stated by the parents that they noticed nothing amiss with their baby just prior to death), and as some of the infants are found dead face downwards or beneath the bed clothes, the sudden death is attributed to accidental suffocation. In addition, inspection of the baby in his bed just after death may show considerable cyanosis, especially of the ears, face and lips, further suggesting that death has occurred from suffocation.

However, careful inquiry shows that many of these children are not in perfect health before death, and also shows that, because of the position of the baby in some of these cases in which the nose and mouth have not been occluded by the bedding, some other cause must be looked for to account for the death. This is illustrated by the following history.

A female child (Case 3), aged twenty-two days, was a little under weight. She had not taken her food well since birth, and had not made the progress that was expected of her. (This history was obtained some time after the death occurred.) Nevertheless, it was thought that there was nothing seriously wrong by those who were in charge of this infant. On the afternoon of her death she was picked up and fed, when she appeared to collapse. She was then put to bed on her side, and when the doctor arrived she was dead—an entirely unexpected death. Post-mortem examination revealed typical tuberculosis of the lungs (Figure I).

In another instance, a baby of five weeks was found dead face downwards in his cot at 6 a.m. He had cried at 2 a.m., when he was given attention and placed on his side. The baby showed the usual post-mortem findings of an asphyxial mode of death, and it was concluded that he had suffocated.[1] Later, when the parents had recovered from the shock of the infant's death, it was stated that this baby had been unusually quiet for forty-eight hours and extremely drowsy for twenty-four hours prior to his death—he wanted to sleep all the time; this evidence suggested that the baby was in reality suffering from some undiscovered natural disease.

When such a death occurs, the parents are as a rule so distraught that a reliable history is not obtained, but sometimes afterwards the parents can point to something which indicates that the baby was not in perfect health, although they stated that he was at the time. On looking back over the evidence given in our previous cases of accidental suffocation in Melbourne, one is impressed by the frequency with which the deceased children did have some symptoms prior to death suggesting that they were suffering from some natural disease.

[1] No microscopic investigation carried out.

One morning, some three years ago, two babies were brought to the morgue, having been found dead in their cots. They came from suburbs twenty miles apart, and inquiry elicited the fact that in both houses the remaining children in each family were suffering from measles. Although in both these cases nothing of note was found at autopsy to account for the deaths, the history strongly suggested death from natural disease.[1]

Rapid changes may take place in the body tissue in disease. Cellular changes may be advanced in a few hours. This may be illustrated as follows. From the edge of a clean surgical incision made on the abdomen, a thin slice of tissue was cut fifty minutes later. Examination of a microscopic section revealed an accumulation of inflammatory cells; there were polymorphonuclear cells and macrophages in the subcutaneous tissue; the capillaries were dilated, and one was stuffed with polymorphonuclear leucocytes.

Such rapid changes may lead to sudden death in infants with little or no evidence of illness prior to death.

FIGURE I.
Pulmonary tuberculosis in a baby, aged twenty-two days (Case 3). Caseation, giant cells and fibrosis present. (High power.)

A child, aged two years, wakened his parents about midnight with stridor. A doctor was called, who treated him in the usual way. Two hours later the doctor was called again—the child was dead. Autopsy later that morning revealed redness and swelling about the larynx, with the usual appearance of a suffocative death. Examination of microscopic sections of the epiglottis revealed an intense acute inflammatory response; the tissues were engorged and literally packed with cells.

An infant, aged three years, was said to have had two epistaxes and was admitted to hospital, where he was given a blood transfusion. Sixteen hours after his admission to hospital, death occurred. Autopsy revealed intense œdema of the lung and considerable congestion of the brain. Microscopic investigation revealed poliomyelitis affecting the brain stem; cellular infiltration had occurred, and perivascular cuffing was pronounced.

Œdema of the lung makes one always pay special attention to the brain; it led us to the diagnosis in unsuspected bulbar poliomyelitis in three recent cases.

A child, aged twenty months, thought to be well, vomited and died suddenly whilst he was being fed. It was thought that this baby might have suffocated from inhalation of vomitus—a supposition which gathered some strength from the autopsy findings, because vomitus was found in the bronchi and bronchioles. Autopsy did not reveal to the naked eye any abnormality of note, apart from a little dilatation of the left ventricle and pallor of the cardiac muscle. Microscopic examination revealed acute interstitial myocarditis. Examination of the heart muscle revealed patchy necrosis, and the muscle was heavily infiltrated with mononuclear and polymorphonuclear cells. Toxic spoiling was apparent in the small vessels.

Acute interstitial myocarditis was found also in Cases 1 and 17. In the latter case the doctor who examined the child assumed that the baby asphyxiated from vomitus and was prepared to give a certificate to that effect.

These cases illustrate three important points. Naked-eye examination may fail to indicate a fatal pathological change in organs. The presence of vomitus in the bronchial tree of itself should not lead to the conclusion that the inhaled vomitus was responsible for the death of the subject. A baby may be thought to be well (even by trained observers), and yet he may be suffering from a fatal illness.

FIGURE II.
Acute myocarditis in a baby found dead in bed face downwards (Cases 1 and 17). (High power.)

One further case emphasizes another point of interest.

A child of three years, thought to be well, whilst playing in the street collapsed and died. Autopsy revealed tuberculosis of the upper lobe of the right lung. There were caseous glands near the bifurcation of the trachea. A caseous gland had eaten its way through the wall of the trachea—suddenly it discharged its contents into the trachea, causing death from asphyxia.

Thus, although we describe the death of a child as sudden, serious undiscovered disease may be present for some time prior to death.

To sum up, a baby may be overwhelmed in a few hours by disease; or on the other hand, although he dies suddenly, he may have been suffering from natural disease and it may not have been realized that he was ill.

It is interesting to pause for a moment to consider how a normal baby sleeps in his bed. These remarks are based on observations made on approximately 100 babies in three separate institutions. It appears that a natural

[1] No microscopic investigation carried out.

sleeping position of a very young baby (less than six months) is flat on the back with the arms raised above the head, lying on the mattress in the crucifix position. When a young baby (less than six months) is placed in the usual position adopted—that is, on his side with the lower shoulder a little in front of the rest of the body—his natural desire appears to be to turn onto his back to sleep in the crucifix position. If these young babies can free their hands and arms, they like to throw them up above the head. Table I shows the sleeping position adopted by some infants in two institutions. The young babies were put to bed on the side, wrapped up with their arms inside their comfort rugs. Nearly all favoured the dorsal position with the arms up above the head.

Some babies at the age of four months can turn over. When a baby can freely move about, his natural tendency appears to be to turn over onto his face to sleep. It will be found that a great many babies of eight months and over adopt the nearly prone position to sleep. Some of

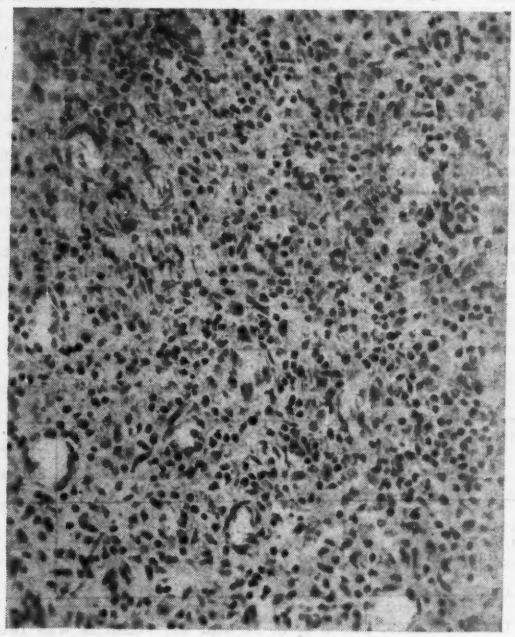

FIGURE III.

Widespread inflammation of the renal medulla (Case 22). It has the features of a subacute lesion. Baby dead, head covered with a blanket. (Also seen in Case 40.) (Low power.)

these older babies will be found face downwards on the mattress, others prone with the face turned to one side, and others nearly prone with one side of the body raised a little off the mattress and the face turned to that side. These babies like the flexed position with the knees drawn up and the body nearly prone. If, then, this is the position of choice of many of the older babies, and if some of them should die of unsuspected natural disease, it may be anticipated that some babies will be found dead face downwards and in a pillow if there happens to be one. This is emphasized by cases listed below.

The custom of placing a baby to sleep on his side is partly directed towards facilitating the exit of vomitus from the mouth should he vomit or regurgitate some of his food. It may be remarked in passing that suffocation from inhalation of vomitus in a healthy baby in my experience is a rarity. When one considers that the babies in Melbourne must vomit literally thousands of times in a year, and that not one case occurred in this city in the last twelve months of death from inhalation of vomitus, either the usually adopted lateral sleeping position for babies has dealt a fatal blow to inhalation of vomitus as a cause of death, or else it never was a serious practical risk.

That is not to say that vomitus is not found in the air passages in some of the babies found dead in bed. Vomiting is a common terminal event or part of the death process when a person dies from various causes. A thorough attempt must be made at autopsy to exclude natural disease before concluding that a baby has died from inhalation of vomitus.

In a sense, a healthy baby can take care of himself, and if ordinary care is taken in putting the baby to bed, there is, in my opinion, virtually no risk of accidental suffocation from the bed clothes. The following observations support that statement.

A healthy baby, aged twenty-six days, asleep in the crucifix position, had a blanket placed over his face. He soon began to stir and rotated his head from side to side,

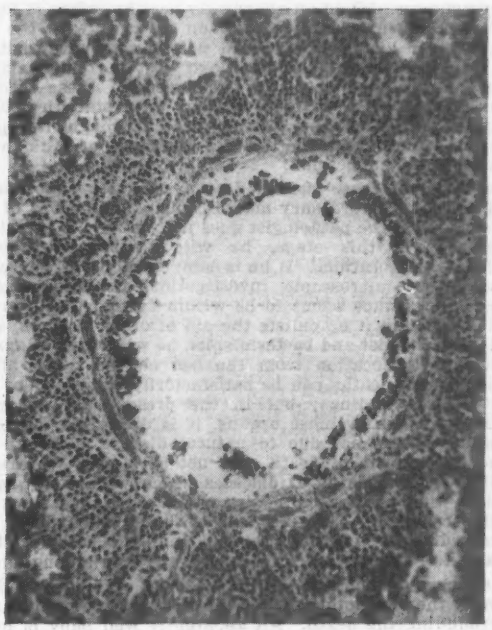

FIGURE IV.

Bronchiolitis (Case 36). Baby dead under the bed clothes. The cells are mostly macrophages; the lesion is probably due to a virus infection. (Medium power.)

finally coming to rest with his head turned well round to the right where he appeared to be content.

A six-weeks-old baby, placed face downwards on a mattress, turned his head freely from side to side, and was quite able to clear his nose and mouth away from the mattress which, in this case, was an institutional one and fairly firm.

A fifteen-weeks-old baby was completely enveloped in a blanket which was drawn firmly around him; he was then placed face downwards on a mattress. He first turned himself over onto his back, then brought his hands forwards in front of his face, pushed the blanket away to give himself breathing space, and whilst still covered, proceeded to suck his thumb and go to sleep.

A four-months-old baby, sleeping on his back, had a blanket placed over his face and head. Almost at once, he raised his hands and flung the blanket down onto his chest.

It has always seemed unreasonable to some observers that a healthy baby should not make a vigorous attempt to save himself should his face become covered with bed clothes, or should he accidentally turn over onto his face. Why should not a healthy baby cry out to attract the

attention of his mother, who is usually sleeping with her ears open, to waken almost the moment the baby stirs?

Two instances were brought to my attention in which young babies who were able to crawl about in bed awakened the parents night after night by screaming. Put to bed in the usual position, they had crawled down to the bottom of the bed under the bed clothes, and finding themselves imprisoned, proceeded to "yell the place down" until released.

Why should a healthy baby just die without much fuss, because he happens to be face downwards or because he has his face covered with bed clothes? But a baby dying of natural disease might well be expected to make a quiet exit.

Our past statistics on accidental suffocation are unreliable, and have erred on the side of gross exaggeration. In Victoria, 24 babies are recorded as having accidentally suffocated in 1947. Sixteen are said to have so died in 1948. In Melbourne in the past twelve months, no babies were accidentally suffocated by bed clothes. Accidental suffocation should never be concluded on circumstances. When an apparently healthy infant is found dead face downwards in bed, or on his back with the bed clothes over his face, it should not be concluded that he has suffocated without a thorough post-mortem examination. Suffocation has been concluded in these circumstances, and this is one reason why statistics over-exaggerate the position. The more thorough the post-mortem examination, the less the likelihood of a diagnosis of accidental suffocation.

In many of these sudden deaths, no gross abnormality will be found on ordinary macroscopic examinations of the organs, and if the pathologist does not pursue his investigations beyond this stage, he will diagnose too many accidental suffocations. If he is content only with thorough post-mortem microscopic investigation, including organs whose appearance seems to be within normal limits to the naked eye, and if he enlists the aid of other workers, such as the biochemist and bacteriologist, he will rarely diagnose accidental suffocation from the bed clothes. Not all of these sudden deaths can be satisfactorily explained by the post-mortem findings; but in the presence of abnormal findings in one or more organs, it is difficult to conclude that death was not due to natural disease, although the exact mechanism of death may not be clear. We are all still probably looking at diseases which we are not recognizing—they have yet to be described. One is not surprised that occasionally cases are met with in which death has occurred from natural disease and the pathologist is unable to recognize it. This is not rare even among adults.

On what evidence is the diagnosis of accidental suffocation by the bed clothes made? There are the circumstances surrounding the death. An apparently well baby is found face downwards dead in his bed. He is livid. There is perhaps pallor about the face. The blood is dark and fluid. The heart on the right side and the veins may be full of dark fluid blood. There are showers of petechiæ on various organs, which are generally congested. There may be a little froth in the bronchial passages. That is a picture found in suffocation, but it is found in disease states as well, and the diagnosis of accidental suffocation must not rest on that alone. There is no autopsy finding in any of the organs that is pathognomonic of accidental suffocation—it is not an organic diagnosis. The above picture when found indicates that the mode of death is asphyxial, but it does not follow that the cause is accidental suffocation.

In Table II are set out some unselected autopsy cases of babies aged less than one year in which death was sudden. The series is consecutive. In nearly all instances the baby was found dead in bed. Only the salient features are listed. In some of the cases the investigation carried out was not as complete as it might have been; nevertheless the analysis speaks for itself.

In some of these cases, the exact position of the child when found could not be ascertained. The parents were so upset that they could not clearly recall the position of the baby, and when a doctor arrived, it was doubtful whether the position in which he saw the child was the position in which death occurred. This means that the pathologist has to allow for the fact that the child may have been face down when found.

In Case 1, the child was dead face downwards, which suggested suffocation, because the parents thought the baby was perfectly well. Post-mortem examination revealed enlarged firm mediastinal glands and Peyer's patches that were prominent and hyperæmic. The heart muscle was pale. Routine microscopic examination disclosed acute bronchitis and acute interstitial myocarditis. The myocardium was heavily infiltrated with cells. Examination of the lymph glands near the heart revealed hyperæmia and early acute inflammation. The microscopic appearance in the heart was similar to that of the baby mentioned who died while being fed, and also similar to that found in Case 17 (see Figure II). Even if no particular lesion had been detected microscopically, considering the fact that the child was eleven months of age and knowing that a child of that age can turn himself about, one should still hesitate to call the cause of death accidental suffocation.

Case 3 is of much interest, in that microscopic examination of the section of lung revealed a typical tuberculous lesion.[1] There were giant cell systems, there was caseation, and there was some fibrosis. An interesting feature was the degree of capillary vascularity at the margins of some of the tuberculous areas. It is stated that the incubation period for tuberculosis in humans is about thirty days. The interesting question arises—is this an example of congenital tuberculosis? The mother was said to be well; she had six other children and had not herself suffered from any chest complaint; she was symptom-free. The child was illegitimate; the mother's history was taken by a nurse. The mother stated that she had known the father for six years and he was not suffering from any known disease. The circumstances surrounding the birth of this child made it difficult for any further investigation of the parents.

TABLE I.

Infants Aged Two to Seven Months, Put to Bed Lying on the Side (19).		Infants Aged Seven to Eighteen Months (34).	
Sleeping Position Found.	Number of Subjects.	Sleeping Position Found.	Number of Subjects.
On side	2	On back	8
On back:	17	Face downwards ..	26
Hands up .. 13			
Hands out 4			

In four cases, Cases 4, 8, 11 and 27, vomitus was found in the bronchial tree, but this was considered to be a terminal manifestation and not causative of the death. Patent *foramen ovale* was noticed in ten instances, but by itself was not considered of great significance. It is said that on an average the *ductus arteriosus* closes in seven to ten days (this is variable); but in four of these cases the ductus was abnormal in that it was patent, but not widely so. In some children the ductus closes slowly; there may be a slight channel up to the age of six months.

In two cases extramedullary hæmatopoiesis was noticed. In addition to the hæmatopoiesis in Case 7, there were pronounced changes in the liver. There was necrosis of liver cells; there was phagocytosis by the Kupffer cells; and there were large numbers of polymorphonuclear cells scattered throughout the liver with no particular relationship to the portal canals.

In Case 12, pronounced changes were found in the liver, with much destruction of its tissue and with fibrous tissue between the cells. This appearance was not complicated by the Rhesus factor. The mother of this child was Rh-positive and also reacted to the Wassermann test.

Case 18 is an unusual example of cardiac abnormality. The right ventricle and the right auricle were grossly dilated and hypertrophied, and the tricuspid valve was incompetent. The valve cusps appeared normal, and no

[1] Reaction to inhaled material excluded on histological grounds.

TABLE II.

Case Number.	Sex.	Age.	How Found.	Macroscopic Findings.	Microscopic Findings.	History.
1	M.	11 months.	Dead face downwards in cot.	Enlarged mediastinal glands. Hyperæmic prominent Peyer's patches.	Acute interstitial myocarditis. Acute bronchitis (Figure II).	Thought to have been well.
2	F.	2 weeks.	Dead in bed with mother.	Heart—patent *foramen ovale* and *ductus arteriosus*. Left preauricular defect. Left *otitis media*.	Lungs—patchy collapse and pneumonic consolidation. Extramedullary hæmatopoiesis of liver.	Mother says the child was born with bronchitis and jaundice. Cough and nasal discharge for some days before death.
3	F.	22 days.	Lying on side in cot.	Petechiæ on heart, lungs and thymus. Patchy consolidation in lungs.	Tuberculosis of lungs (Figure I).	Illness not suspected. Appeared to collapse when last fed.
4	M.	7 weeks.	Dead in bassinette when doctor saw him.	Patent *foramen ovale*. Trachea and bronchi contained vomitus.	Acute enteritis with ulceration.	Sickly since birth. Gastro-enteritis three weeks before death.
5	M.	14 weeks.	Dead face downwards in cot.	Patent *foramen ovale*. Redness and œdema about larynx.	Early bronchopneumonia. Epiglottis — superficial inflammatory ulceration.	Left on back fifteen minutes prior to death. Thought to be well.
6	M.	10 weeks.	Lying in cot, position not stated.	Congestion of mucosa of trachea and bronchi.	Early acute inflammatory changes in lung and pleura.	Seen three hours before; thought to be healthy.
7	M.	2 months.	Dead in cot, position not stated.	Skin jaundiced. Patent *foramen ovale*. Enlarged firm spleen. Ascites. Right-sided hydrocele.	Extramedullary hæmatopoiesis, liver, spleen, *plus* hepatitis.	Weighed three pounds at birth, two months premature. Mother died of cerebral hæmorrhage.
8	F.	6 months.	Lying in perambulator on side, neck resting across restraining plastic harness.	Vomitus in bronchial tree.	Congestion of meninges with an increase of lymphocytes. Bronchitis and bronchiolitis.	Had a cold for two days prior to death.
9	F.	10 weeks.	Face downwards in perambulator, face in pillow.	Bronchopneumonia.	——	Child was considered to have respiratory infection before death.
10	F.	40 hours.	Lying in cot on back.	Torn *tentorium cerebelli*. Intracranial hæmorrhage. Prematurity.	Primary atelectasis.	Premature—about 32 weeks.
11	M.	4 months.	Face downwards in cot.	*Otitis media*. Terminal vomiting.	Acute bronchitis.	Fifteen minutes before death was lying on back, apparently well.
12	M.	1 month.	Lying in bassinette, position not stated.	Jaundiced. Spleen firm and slightly enlarged. Irregular femoral epiphysis.	Syphilitic hepatitis.	Jaundiced since birth. Mother Rh-positive and reacted to Wassermann test.
13	F.	10 weeks.	In double bed with parents. Mother wakened by baby making gasping noises at death.	Weight four pounds five ounces. Gross congenital heart disease.	No microscopic examination made.	Three hours previously child's crying wakened parents; napkin was changed and she went to sleep.
14	M.	10 weeks.	Found in convulsions; dead when doctor examined him.	Right *otitis media*. Multiple petechiæ.	Lung, œdema. Some areas of collapse. Acute bronchitis and bronchiolitis.	Parents thought child had a rise in temperature on evening before death. Attributed to teething.
15	M.	5 months.	Lying on back in double bed with parents. Not covered with bed clothes.	Bronchopneumonia.	No microscopic examination made.	Subject to "colds" since birth.
16	F.	8 months.	Face downwards in cot.	Bilateral *otitis media*.	Acute bronchitis and bronchiolitis — probably virus infection.	Usually slept face down. "Bronchitic" cold one week before—listless in interim.
17	M.	12 days.	Vomited; dead when doctor arrived.	Dilated pale heart. Enlarged gland at tracheal bifurcation.	Acute interstitial myocarditis with considerable necrosis of muscle. (Figure II.)	Off food before death. Assumed to have asphyxiated from vomitus.
18	F.	3 weeks.	Child stiffened and died whilst being nursed by mother.	Great hypertrophy of right auricle and ventricle. Patent *foramen ovale*.	Heart, hypertrophy of muscle fibres. Some thickening of pulmonary arterioles.	No previous history.
19	F.	11 days.	Died soon after arrival at hospital.	Malnutrition. Patent *foramen ovale*.	Heart, glycogen storage disease (von Gierke). (Figure V.)	Two weeks premature. Weight lost rapidly after birth. A "difficult feeder".
20	M.	2 days.	Lying on left side in cot.	Primary atelectasis.	Atelectasis.	Baby appeared well prior to death.
21	M.	5½ months.	Lying in cot, position not stated.	Right *otitis media*. Infection in scalp.	Acute bronchitis and bronchiolitis.	Suffered from eczema for some weeks before death.
22	M.	11 weeks.	Lying on right side, dead in perambulator, two pillows under head. Blankets over head.	Frothy fluid in air passages. Areas of bluish-red discoloration throughout kidneys.	Extensive inflammatory changes in kidney, medulla and cortex. Subacute nephritis. (Figure III.)	Child "off colour" for five days before death. In hospital during previous month for malnutrition.
23	M.	2½ months.	Lying in cot, position not stated.	Patent *foramen ovale* and *ductus arteriosus*. Hypertrophied left ventricle. Brownish discoloration in subarachnoid space over right hemisphere.	No microscopic examination made.	Put to bed eight hours before, apparently in good health.
24	F.	5 months.	Face downwards in cot on mattress. Flock pillow.	Multiple petechiæ on heart and lungs.	Acute bronchitis and bronchiolitis. Gross œdema of lung.	Awakened crying four hour before, but thought to be well.

TABLE II.—*Continued.*

Case Number.	Sex.	Age.	How Found.	Macroscopic Findings.	Microscopic Findings.	History.
25	M.	7 days.	Face downwards in cot.	Infected umbilicus (sufficient to prove fatal).	Lungs, patchy atelectasis. Great thickening of inter-alveolar walls and vessels with endarteritis.	Lying on back five minutes before death, apparently well.
26	F.	10 weeks.	Lying on back in bassinette. Baby's bottle in position, teat still in child's mouth.	Hæmorrhages on lungs. Patchy consolidation.	Acute bronchitis and bronchiolitis.	No evidence of illness.
27	M.	11 weeks.	Dead beneath blankets in cot; head covered.	Vomitus in bronchi.	Acute bronchiolitis. Many polymorphonuclear leucocytes in walls.	Had a "cold" for fourteen days.
28	M.	18½ months.	Lying on left side in cot. Face and lips cyanosed.	Patent *foramen ovale*. Bilateral *otitis media*. Bronchiectasis (upper lobe of right lung).	Lipoid pneumonia. Bronchiectasis with considerable lymphoid hyperplasia.	Frequent attacks of pneumonia. Child was a Mongol.
29	M.	2 months.	Dead in perambulator, position not stated.	Some hyperæmic areas in small intestine.	Enteritis and patchy necrosis of liver cells.	Abscess on nose three weeks before death.
30	F.	8 weeks.	Dead on back in her cot. Face clear.	Nothing definite.	Bronchitis and bronchiolitis. Alveolar œdema.	Was not well four hours before death; refused her bottle.
31	F.	7 months.	Died in mother's arms.	Redness and œdema of larynx. Diffusely pink brain.	Inflammatory ulceration of epiglottis.	——
32	M.	8 months.	Dead when first examined by doctor.	*Otitis media*. Patent *foramen ovale*. Large blotchy rash on body. Bronchopneumonia.	Bronchopneumonia. Meningo-encephalitis.	Had a slight cold for one week before death.
33	M.	19 days.	Dead face downwards in perambulator.	Heart a little enlarged. Patent *ductus arteriosus* and *foramen ovale*. Considerable atelectasis.	No microscopic examination made.	No illness.
34	M.	10 months.	Dead when taken to hospital.	Purulent meningitis, mostly on frontal lobes.	No microscopic examination made.	Temperature 103° F. on previous evening, with stiffness of arms and legs.
35	F.	6 weeks.	Dead when examined by doctor.	Diffuse subarachnoid hæmorrhage. Multiple hæmorrhages about body. Purpura.	No microscopic examination made.	Bruised easily since birth. Increased platelet count when previously in hospital.
36	F.	9 months.	Dead in bed beneath blankets. Head covered.	Hæmorrhages on lungs and thymus.	Bronchiolitis (see Figure IV). Probably virus infection.	Slight cold for two days before death. Thought to be teething.
37	F.	5 weeks.	Dead in perambulator, face clear of bedding, lying on side.	Atelectasis of upper lobe of right lung.	Pneumonic consolidation of upper lobe of right lung and early meningitis.	Thought to be well the night before when put to bed. Cried during the night. Found dead at 8 a.m.
38	F.	7 months.	Face downwards in a pillow.	Very pink brain.	Acute bronchiolitis. Œdema of lung.	Thought to be well.
39	F.	7 weeks.	Face down in perambulator.	Nothing definite, apart from asphyxial mode of death.	*Periarteritis nodosa*. Acute bronchitis and bronchiolitis.	No illness. Left on back two hours before.
40	F.	5 months.	Dead in cot, position uncertain.	Under-nourished. Some vomitus in air passages.	Subacute nephritis. Thickened meninges (with fibrosis.) (Figure III.)	Ten weeks premature. "Off food" before death.

abnormality was found on microscopic examination of the heart apart from hypertrophy of the fibres. Some thickening of the pulmonary arterioles was present.

Case 22 presents an unusual lesion (Figure III). The greater part of the medulla and much of the cortex of the kidney were involved in a subacute inflammatory process. The lesion consisted of focal destruction of tubules and widespread infiltration of the interstitial tissue with lymphocytes, plasma cells, endothelial cells, some polymorphonuclear leucocytes and fibroblasts. There were collections of lymphocytes and polymorphonuclear cells in some of the glomeruli. The picture was that of subacute interstitial nephritis of unknown ætiology. Case 40 was similar.

Case 36 is interesting in that the baby was found dead, completely covered over by blankets. The parents of this baby, when they found her under the blankets, were quite satisfied that she had accidentally suffocated. At the autopsy there was nothing much to see on naked-eye examination, apart from findings which could have been interpreted as due to suffocation. Microscopic examination of all the organs showed that the baby had a pulmonary infection—one of the bronchioles is shown in Figure IV. Bronchiolitis is present; the wall is heavily infiltrated with cells which are nearly all macrophages; a few polymorphonuclear cells were present and there was accompanying capillary dilatation. Examination of the surrounding alveoli revealed little change. This type of lesion escapes the naked eye. Macrophages may be the first on the scene in inflammatory lesions, and in early lesions they are sometimes the only cells present. In very early lung lesions minor degrees of the appearances shown in Figure IV may be seen; there may be only a sprinkling of mononuclear cells in the bronchiolar walls.

There is another aspect of the histological appearance that deserves attention: the appearance illustrated in Figure IV is much the same as that seen in influenza virus infection in mouse lung, in which histologically there is a comparable picture of infiltration of the bronchial wall with monocytic cells, but without spread to the lung parenchyma. It would seem from comparison with studies on mice that Case 37 is an example of a virus infection.

Workers in this field emphasize the slightness of the changes that may be found in the pulmonary tree in young babies who succumb to respiratory infections. In the case illustrated, the baby was known to have had a slight cold for two days, but this was attributed by the parents to "teething". Much attention should be paid to the bronchial tree in these cases of sudden death among young babies, and blocks of tissue should be examined from various parts of the lungs.

Case 19 presented a most interesting finding. The histological appearance of the heart muscle is shown in

Figure V. It is thought to be an example of glycogen storage disease (von Gierke). The muscle cells were vacuolated; the nuclei were often peripheral, the vacuolated appearance making a striking picture. That the cells were filled with glycogen was shown by staining a paraffin section with Best's carmine. The heart was not enlarged and the condition was unsuspected at autopsy. Slight changes were seen in the epithelial cells of the renal tubules and in the liver. This baby was two weeks premature and he had lost weight rapidly after birth and had been very difficult to feed. Although the autopsy was conducted twenty-four hours after death, and although the tissues were formalin hardened and paraffin sections were prepared, the appearance with Best's carmine stain was conclusive.[1]

In Case 39, in which a seven weeks old baby was found dead face downwards, cellular infiltration was present in the kidney, liver and pancreas. This occurred chiefly about

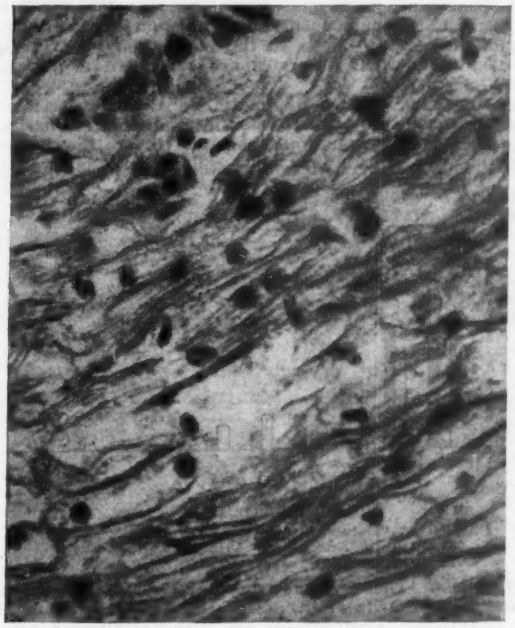

FIGURE V.
Myocardial changes. Glycogen storage disease (von Gierke) (Case 19). (High power.)

blood vessels affected by arteritis with perivascular accumulations of cells—probably *polyarteritis nodosa* (see Figure VI).

Apart from sections of all organs in these cases, one would like to have available the results of a blood culture, a blood smear examination, a white cell count, and a blood sugar estimation *et cetera* made close to death; but these are extra aids which were not available in the cases under discussion.

In ten instances in the series reviewed the baby was found dead face downwards, and in three other cases babies were found dead with the head covered by blankets. In seven of the cases the position of the baby when found was not known. In three other instances the baby was in a double bed with parents. So that out of this total of 40 cases, a diagnosis of accidental suffocation might have been made in 24.

In many of these cases it was assumed before autopsy that accidental suffocation had occurred, because there was no history of illness, or because the child was found lying dead face downwards, or because when the child was viewed by the medical attendant cyanosis was observed as a prominent feature. The autopsy findings in this series of cases make it extremely difficult for the conclusion to be drawn that one of these babies had been accidentally suffocated by bed clothes. Although in every case the pathological findings described do not completely answer the question why sudden death occurred, and although the exact mechanism of death is obscure in some cases, in practically every case natural disease was present. One is impressed on looking over these cases with the frequency with which, on careful inquiry, some evidence of illness was present prior to death.

Lymphoid hyperplasia or prominence is the almost invariable finding in these infants, as it is in babies dead from all manner of ills. It was no more prominent in this series of cases than it is in infants who have died a

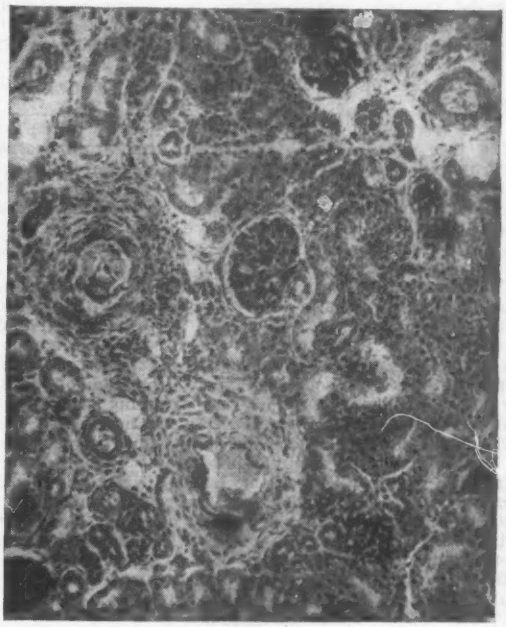

FIGURE VI.
Arterial changes in kidney (*periarteritis nodosa*). Baby of seven weeks found dead face downwards. (Medium power.)

violent death. The thymus is nearly always prominent (unless there has been some active disease present for some little time prior to death); the mesenteric lymph nodes are always enlarged. The Malpighian corpuscles of the spleen are conspicuous. Lymphoid follicles in the wall of the intestine are a feature, and not infrequently one sees Peyer's patches standing up above the surrounding mucous membrane. The enlarged thymus does not compress the trachea, and the prominence of the lymphoid tissues in these cases of alleged accidental suffocation is probably of no more significance in the death than it is in the case of a baby who dies in a motor-car accident.

In making these autopsies and in investigating similar deaths, the evidence of natural disease may be easily overlooked. If the autopsy goes no further than macroscopic examination of the organs, then much will be missed. Routine microscopic examination of the organs of the body may reveal disease which is unsuspected on macroscopic inspection, but it may not help much in the diagnosis of septicæmia, or when the death is due to biochemical changes.

There is a stigma for the parents about death from accidental suffocation. The thought of it may have a

[1] An appearance resembling von Gierke's disease is seen in papillary muscles and deep layer of the myocardium normally in full-time babies.

serious effect on the mother. In one of the cases in which accidental suffocation had previously been concluded by me, the mother was so affected that she was afraid to go out on the street lest other women should say something implying maternal carelessness—she wanted to remove herself from the district. She will carry the scar to the grave. Another mother committed suicide.

In a recent "digest", there was an article for the public in which it was stated that "30,000 babies die each year in America from accidental suffocation". There has been formed a society for the prevention of accidental suffocation. One feels tempted to suggest that if there was a society for the performance of complete autopsies these figures would be greatly reduced. A coroner with wide experience in a large city, in conversation on this subject, said that he was often nonplussed at inquests into these deaths, because where it was alleged on the post-mortem findings that accidental suffocation had occurred, the baby had sometimes been found on his back with no bed clothes over his face. In some cases in which it was pointed out at the inquest that the baby was on his back when found, it was suggested that the weight of the bed clothes on the chest was probably the responsible factor.

Conclusion.

The fear of accidental suffocation causes unnecessary maternal anxiety and gives rise to a great deal of overfussiness on the part of the parents. A doctor recently said that he was having a bad time, because there was a new baby in the house, and his wife was in and out of bed every five minutes during the night to make sure that the baby was not suffocating. It is all so unnecessary.

There should be no departure from the high standard reached and the usual ritual observed in putting a baby to bed. If there must be a pillow, let it be placed under the mattress to raise the baby's head. A firm mattress, the bed clothes firmly stretched across it and tucked well under the sides, is good practice. The custom of pinning the baby's hands by his sides and wrapping him up in a comfort rug so that he cannot free his hands lest harm befall him, does not appear to have much to recommend it—certainly not the fear of suffocation.

In spite of carefully putting babies to bed and placing them on the side or on the back, there will continue to be instances in which babies are found dead face downwards in their cots; but few, if any, will be accidental suffocation in my opinion, but instances of the appalling swiftness with which death in the form of natural disease snatches young life.

Acknowledgements.

I am indebted to Professor P. MacCallum and to Dr. R. Webster, of the Children's Hospital, Melbourne, for their assistance in checking the microscopic findings in many of these cases. At the City Morgue, Dr. R. J. Fleming supplied me with his findings in several of these cases. I am also indebted to Dr. E. Bate and Dr. J. Perry for their assistance with some of the work, and to Dr. E. H. Derrick, of Brisbane, for his help. To the technician at the City Morgue, Mr. H. Quigley, I am greatly indebted for innumerable excellent microscopic sections.

INFANT MORTALITY IN NEW SOUTH WALES.[1]

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THE trends in the mortality rates in the first twelve months of life may be regarded as a measure of infant welfare and a reflection of the standard of living in the community.

[1] Read at a meeting of the Section of Paediatrics, Australasian Medical Congress (British Medical Association), Sixth Session, Perth, August, 1948.

Infantile mortality rates are almost universally calculated by relating the number of deaths at ages under one year in any period to the number of live births in the same period, and are expressed per 1000 live births. The number of live births is taken as the "population exposed to risk". This simple method is regarded as giving a reasonably precise measure of mortality without involved calculations for determining the true number of children from which any group of deaths was drawn.

The practice of tabulation of births and deaths with reference to time affects the presentation of statistics. Some countries tabulate by the date of the event, others by the date of registration. The latter procedure is adopted in New South Wales and other Australian States for reasons of simplicity and convenience.

In New South Wales births are registered fairly promptly, the average lag between birth and registration being about fourteen days.

These latter differences of tabulation and calculation are not important in normal times, but become significant only when the number of births fluctuates quickly from period to period. Even so the effects may be regarded as negligible when the rates are calculated as averages over long periods.

A wide range of racial, climatic, social and other factors must be taken into account when international comparisons are being made, but more important still are the differing practices of tabulation and calculation, as well as

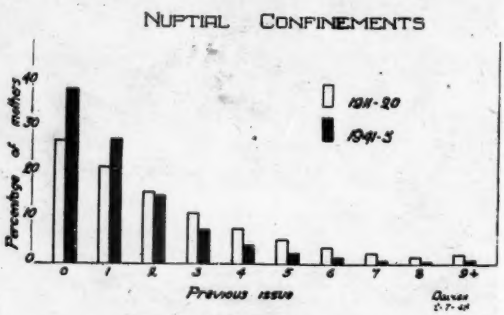

FIGURE I.

Data from "Demography, Official Statistics, Commonwealth of Australia", 1911-1920, 1941-1945.

definition. These become most interesting when comparisons are made of the low rates claimed by a number of countries. Comparisons are also complicated by the inclusion or exclusion of coloured members of the population.

Another important factor in the comparison of international figures is the definition of stillbirth, as differences in this respect affect both the number of live births and the number of infant deaths. In New South Wales the *Registration of Births, Deaths and Marriages Act* defines a stillborn child as follows:

> A "stillborn child" means any child of seven months gestation and over not born alive, and includes any child not born alive which measures at least 14" (fourteen inches), but does not include any child who has actually breathed.

In Holland, another country claiming a remarkably low rate, stillborn children are defined thus: (*a*) children born prematurely and dead; (*b*) full-term children born dead; (*c*) children born alive but dying before registration —that is, within three days—the last-mentioned being registered in the register of deaths with the endorsement "presented dead". (United Nations World Health Organization, 1948.)

The operation of the last category makes any comparison between the figures for Holland and Australia ineffective, because, as will be shown later, the mortality rates are highest in the first week.

In other countries there is divergence of practice, but usually of a minor character, which does not impair valid comparison.

TABLE I.[1]
Infant Mortality, States and Countries.

State or Country.	Deaths under one year per 1,000 Live Births. Average 1941–45	1945
New Zealand	29·53	27·99
South Australia	32·95	28·08
Western Australia	33·30	29·52
Queensland	34·51	29·76
Victoria	34·50	28·03
New South Wales	35·95	30·63
Tasmania	39·31	27·48
Australia	34·97	29·38

State or Country.	Average 1931–35	1943
New Zealand	39	31
Australia	41	36
New South Wales	42	36
Norway	45	*
Netherlands	45	40
Switzerland	48	40
Sweden	50	29
United States	59	40
England and Wales	62	49

Country.	Average 1931–35	1943
South Africa	63	48
Eire	68	80
Denmark	71	45
France	73	75
Germany	74	*
Canada	75	54
Northern Ireland	78	78
Scotland	81	65
Belgium	82	67
Argentina	94	78
Italy	105	*
Spain	113	99
Japan	120	*
Czechoslovakia	130	*
Mexico	134	117
Hungary	157	131
Ceylon	182	*
Rumania	182	*

* Not available.

[1] From the "Official Year Book of New South Wales", Number 50, 1945–1946 (1948).

Infant Mortality Rates of New South Wales Compared with Other Countries and Australian States.

The infant mortality rates of New South Wales compared with those of other countries and Australian States are shown in Table I.

If a comparison of infantile mortality is made on the basis of all infantile deaths, irrespective of colour, Australia has the lowest rate of any country. If the colour factor is eliminated and comparison is made on the white

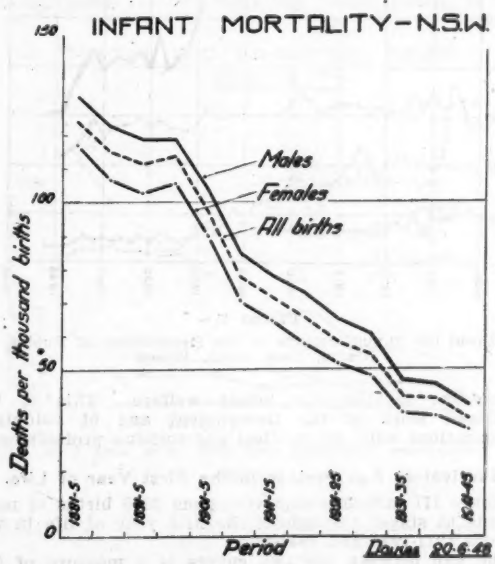

FIGURE II.
Infant mortality rates, New South Wales; from the "Official Year Book of New South Wales", Number 50, 1945-1946 (1948).

population only, New Zealand rates are lowest. The relative number of Maoris and whites in New Zealand are 108,000 and 1,715,000. Compared with this the aboriginal population living in association with whites in Australia is relatively unimportant. For instance, in New South Wales there are only about 500 full-blood aborigines, but on the other hand, half-castes and lesser castes are included with the white population.

Trends in the Size of Families.

The altering pattern of the size of the family is a matter which closely concerns the welfare of infants in the first year of life. Figure I, by showing the nuptial confinements in two different groups of years, illustrates the proportion of mothers bearing children in each in relation to the number of previous issue.

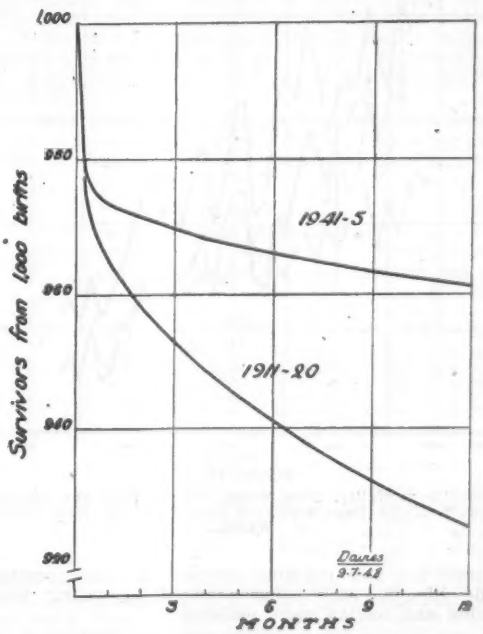

FIGURE III.
Life table, first year of life, males, Australia.

This does not represent the average size of families in the total population; it is a sample composed of married women bearing children. In the total population there is a large number of married women who bear no children.

The figure indicates clearly the approach to the two-child family. Whereas in the past a reasonable proportion of mothers bearing children had already had two or more children, it can be seen in the later period that this proportion had dropped considerably. In the smaller family the reverse is the position.

Although these ratios have changed, as shown, it must be remembered that the figures for the period 1941-1945 embody the war years, in which the normal fertility pattern was disturbed by wartime factors. It may generally be accepted on pre-war experience that the true trend is to the two-child family.

There are, however, optimists among those studying world figures; for instance, the United Nations Statistics Officer (1947) considers that the larger family in western and northern Europe, North America and Australia has come to stay, and bases his claim on the facts that the groups of women who were responsible for the fall of the

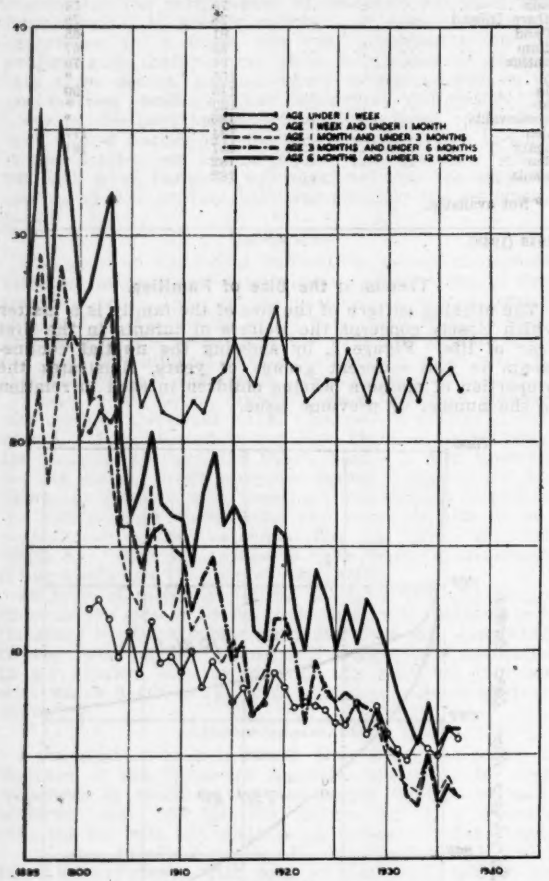

FIGURE IV.

Infantile mortality, New South Wales; from the annual reports of the Department of Public Health, New South Wales.

birthrate are now reaching the end of their potentially fertile life and are being replaced by young women wanting and having more children.

The Reduction in the Infantile Mortality Rate Since 1881.

The year-by-year improvement in the infant mortality rates for New South Wales is shown in Figure II. The points on the curves represent the average of successive five-year periods.

The reduction of infant mortality has continued throughout this century. From a mortality rate of more than 100 per 1000 live births in the first three years, the reduction was rapid and sustained. At the beginning of World War I the rate was 69 per 1000 and ten years later it was 59.

The first time the rate fell below 50 was in 1930. From that time the reduction continued intermittently, the figure varying down to 36 in 1943. From 1944 to 1946 the drop was remarkable, the rate reaching as low as 30 and remaining there. The 1947 figures were below 30—29·82.

These figures are a reflection of the high standard of pædiatric practice and of the progress of infant welfare. In 1904 in Sydney the first health visitor was sent out to visit mothers and advise them on hygiene, and in 1908 the National Council of Women established the Alice Rawson Schools for Mothers. These may be regarded as the forerunners of mothercraft homes and infant welfare centres. In every State the efforts of those interested in the welfare of mothers and babies have resulted in com-

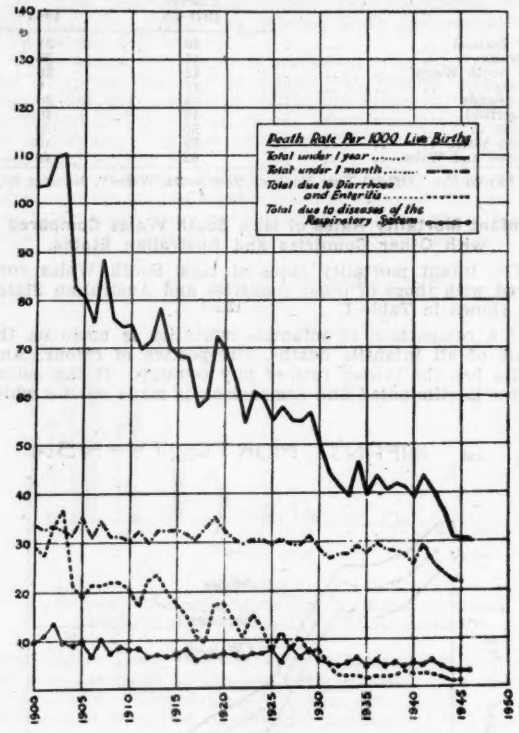

FIGURE V.

From the annual reports of the Department of Public Health, New South Wales.

prehensive services for infant welfare. This is the combined work of the Government and of voluntary organizations with the medical and nursing professions.

Survival at Age Periods in the First Year of Life.

Figure III indicates survivors from 1000 births of male infants at stages throughout the first year of life in the periods 1911-1920 and 1941-1945.

The gap between the two curves is a measure of the saving of infant life, as it shows that many more children now survive who formerly would have died. The flattening of the top curve after the hazards of the first month of life have been overcome shows how the mortality is approaching the irreducible minimum. The curve represents the number of survivors from 1000 born, and if no child died after the first month the curve would be represented by a straight horizontal line from that point.

Masculinity in Infantile Mortality.

The excessively high death rate of males as compared with females is demonstrated throughout the life span. In the first year the male death rate is consistently higher

TABLE II.

Infantile Mortality, New South Wales, According to Age.

Year.	Rate of Mortality per 1000 Live Births among Children.							
	Under One Week.	One Week and Under One Month.	Total Under One Month.	One Month and Under Three Months.	Total Under Three Months.	Three Months and Under Six Months.	Six Months and Under Twelve Months.	Total Under One Year.
1931	21·58	5·22	26·80	4·11	30·91	4·61	8·00	43·52
1932	22·94	4·72	27·66	3·58	31·24	3·07	6·75	41·06
1933	21·90	5·70	27·60	3·42	31·02	2·88	5·45	39·35
1934	24·02	5·12	29·14	4·94	34·08	4·76	7·52	46·36
1935	22·99	4·77	27·76	3·44	31·20	2·87	5·37	39·44
1936	23·64	5·93	29·57	4·07	33·64	3·66	6·17	43·47
1937	22·80	5·58	28·38	3·10	31·48	3·05	6·15	40·68
1938	23·42	4·48	27·90	3·80	31·70	3·46	6·68	41·84
1939	22·96	4·48	27·44	2·48	29·92	3·56	7·54	41·02
1940	21·12	4·46	25·58	3·85	29·43	3·62	5·97	39·02
1941	23·55	5·97	29·52	4·23	33·75	4·18	5·84	43·77
1942	20·97	4·52	25·49	3·97	29·46	4·27	6·46	40·19
1943	19·61	4·23	23·84	3·56	27·40	3·42	5·36	36·18
1944	18·30	3·66	21·96	2·16	24·12	2·38	4·18	30·68
1945	18·28	3·52	21·80	2·43	24·23	2·61	3·79	30·63
1946	18·82	3·14	21·96	2·19	24·15	2·37	3·70	30·22

than the female. The higher male mortality begins in the fœtal stage, as indicated by the high proportions of males in stillbirths.

In New South Wales from 1936 to 1940 the proportion of masculinity in stillbirths was 132·7 males to every 100 females; and in the period from 1941-1945 this was 126·4 to every 100 females.

Mr. W. J. Willcocks, of the New South Wales Bureau of Statistics and Economics, has taken out complete figures for male and female infant mortality for all diseases in each age group for both male and female infants in the

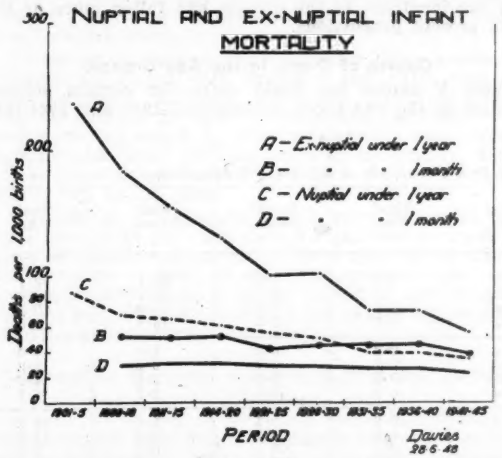

FIGURE VI.

Data from the "Official Year Book of New South Wales", Number 50, 1945-1946 (1948).

first year. A higher rate for males can be demonstrated for every disease with the notable exception of whooping cough, in which the death rate among females in all age groups is higher than the rate among males. In the rates of deaths from the major groups of causes, the masculinity is prominent in prematurity, congenital debility, injury at birth, malformation, respiratory diseases.

Mortality Rates in Relation to Ages in Weeks and Months.

The dramatic reduction that has occurred in the infant mortality is illustrated in Figure IV. It is shown, however, that little or no improvement has taken place in the rate for the first week of life.

Table II shows the tabulated figures in age groups, which highlight in no uncertain manner the contribution to infant deaths of prematurity, malformation, congenital debility and other diseases peculiar to very early infancy. If we are to expect a further reduction in infant mortality, special efforts will be required to prevent many of the deaths from these causes. This means better pre-natal care of the mother. More than that, it means improved obstetrics and better knowledge of the fundamental principles of neo-natal care of the baby and improved nutrition of the mother.

In Sydney premature babies are frequently lost because they arrive at mothercraft homes chilled. The other cause of death of these frail infants is careless handling; even the wrapping of the infant in cotton-wool must be done with knowledge that next to chilling, exhaustion is the most frequent cause of loss of life in this condition.

The provision of special nurseries at large obstetric hospitals is necessary for premature babies, for all babies who are exhausted by long labours or who suffer trauma from instrumental deliveries, and also for those from toxæmic mothers whose vitality is lowered. Attentive skill offered to these infants in suitable surroundings has been proved to save many lives.

TABLE III.

Probability of Dying Within One Year.[1]

	1881–1890	1891–1900	1901–1910	1920–1922	1932–1934
Males.					
q_0	0·1325	0·1184	0·0951	0·0713	0·0454
q_1	0·0349	0·0264	0·0178	0·0146	0·0078
q_2	0·0137	0·0098	0·0068	0·0063	0·0038
q_3	0·0089	0·0063	0·0044	0·0042	0·0029
q_4	0·0069	0·0050	0·0035	0·0030	0·0021
Females.					
q_0	0·1157	0·1014	0·0795	0·0557	0·0364
q_1	0·0335	0·0248	0·0167	0·0125	0·0065
q_2	0·0133	0·0096	0·0063	0·0052	0·0033
q_3	0·0083	0·0062	0·0041	0·0037	0·0024
q_4	0·0065	0·0049	0·0032	0·0030	0·0021

[1] Data from the 1933 Census, Australian Life Tables. Note: qx, as given in life tables, is the probability that a child now at his xth birthday will survive one year, that is, to his $(x+1)$th) birthday. Deaths in the periods given are divided by appropriate population estimates to give mortality rates from which qx is derived.

TABLE IV.[1]

Infant Mortality, New South Wales, Metropolis and Country, 1927 to 1945.

Year.	Metropolis.		Remainder of State.		New South Wales.		Greater Newcastle.	Remainder of State.
	Average Annual Deaths under One.	Rate per 1,000 Live Births.	Average Annual Deaths under One.	Rate per 1,000 Live Births.	Average Annual Deaths under One.	Rate per 1,000 Live Births.	Rate per 1,000 Live Births.	Rate per 1,000 Live Births.
1927–30 ..	970	53·14	1,339	54·78	2,309	54·08	55·44	54·74
1931–35 ..	702	40·11	1,184	43·12	1,886	41·95	48·09	42·80
1936–40 ..	716	38·18	1,247	43·12	1,963	41·18	40·23	43·31
1941–45 ..	848	32·52	1,186	38·87	2,034	35·95	37·36	39·01

[1] From the "Official Year Book of New South Wales", Number 50, 1945–46 (1948).

Maintenance of Improvement of Mortality Rates in the Pre-School Years.

One often hears the contention that many of the lives of premature babies and others saved in the first year of life are not worth saving, as the children remain frail and are likely to die in their pre-school years.

Table III presents figures indicating the continued improvement in the mortality rates of the survivors of the first year and the maintenance of this improvement as the years go on. The figures are the qx figures of successive life tables and they represent the probability of dying within one year.

Comparison of Infantile Mortality Rates in Different Parts of the State.

The trends of infantile death rates in Sydney, in Newcastle, in the remainder of the State, and in the State as a whole, in five-year periods beginning from 1927-1930, are shown in Table IV.

This table commenced at 1927, as from that year births and deaths have been tabulated in New South Wales on the basis of usual residence. It is only since infant mortality rates have been on this basis that they are comparable from area to area. Before 1927 births and deaths were tabulated according to place of occurrence, and the degree of hospitalization invalidated all comparisons of this nature.

Since this change was made in New South Wales, special care has been taken to assign infant deaths to the usual residence of the mothers. In the case of children born in hospital and dying before the discharge of the mother from hospital, the death is given the same residence as that shown for the mother in the birth registration.

These figures show that the City of Greater Newcastle and the remainder of the State have higher mortality rates than Sydney. The comparisons in five-year periods show improvement, but the rates outside the metropolitan area still remain higher than those within. The City of Greater Newcastle shows more improvement than the remainder of the State, the latter rate being the higher.

Death Rates from Respiratory Diseases and from Diarrhœa and Enteritis in the First Year of Life.

Figure V shows the mortality rates due to two major diseases, diarrhœa and enteritis, in which reduction has been pronounced and sustained, and to respiratory diseases which show only a minor reduction. These disease groups are shown in relation to the total death rates and to the rates for ages under one month.

The greatest over-all improvement in rates from all causes is in the decline of diarrhœa and enteritis, which together, during the first quarter of the century, were one of the major causes of death and represented a dreaded community scourge with the return of every summer. By 1933 the incidence of the disease had fallen more or less to its present proportions.

Causes of Death in the Age Groups.

Table V shows the death rates for certain selected diseases in the two broad periods 1911-1920 and 1941-1945,

TABLE V.

Australian Infantile Death Rates from Certain Causes in the First Four Weeks of Life per 1000 Live Births.[1]

Cause.	Period.	Sex.	First Week.	Second Week.	Third Week.	Fourth Week.	First Month.	First Year.
Premature births, injury at birth.	1911–1920	M.	21·2	2·8	1·6	1·1	26·7	32·1
		F.	16·4	2·1	1·3	0·9	20·9	25·3
	1941–1945	M.	17·8	1·4	0·6	0·3	20·0	20·7
		F.	13·7	1·1	0·5	0·3	15·5	16·0
Malformations	1911–1920	M.	2·0	0·4	0·2	0·2	2·8	4·0
		F.	1·5	0·3	0·2	0·1	2·2	3·2
	1941–1945	M.	2·2	0·5	0·2	0·2	3·1	4·7
		F.	2·0	0·4	0·2	0·1	2·7	4·1
Diarrhœa and enteritis ..	1911–1920	M.	0·1	0·3	0·3	0·4	1·1	18·2
		F.	0·1	0·1	0·2	0·2	0·7	14·9
	1941–1945	M.	0·0	0·1	0·1	0·1	0·3	2·1
		F.	0·0	0·0	0·1	0·1	0·2	1·5
All pneumonia, bronchitis *et cetera*, excluding influenza.	1911–1920	M.	0·2	0·3	0·3	0·3	1·1	6·6
		F.	0·1	0·2	0·2	0·2	0·8	5·5
	1941–1945	M.	0·4	0·3	0·3	0·2	1·1	4·6
		F.	0·4	0·2	0·2	0·2	0·9	3·9
Convulsions	1911–1920	M.	0·8	0·2	0·1	0·1	1·4	2·3
		F.	0·5	0·2	0·1	0·1	0·9	1·7
	1941–1945	M.	0·1	0·0	0·0	0·0	0·1	0·2
		F.	0·0	0·0	0·0	0·0	0·0	0·1
Meningitis (meningococcal) ..	1911–1920	M.	0·1	0·0	0·0	0·0	0·2	1·8
		F.	0·1	0·0	0·0	0·0	0·1	1·4
	1941–1945	M.	0·0	0·0	0·0	0·0	0·0	0·3
		F.	0·0	0·0	0·0	0·0	0·0	0·2
All other causes	1911–1920	M.	1·0	0·6	0·6	0·4	2·4	9·7
	1941–1945	F.	1·0	0·6	0·4	0·4	1·8	8·0
		M.	1·2	0·2	0·2	0·4	2·1	6·2
		F.	0·9	0·3	0·1	0·1	1·6	5·1

[1] Some of the rows, especially in "All other causes", do not balance exactly because of an accumulation of "rounding off" figures.

TABLE VII.

The Months of Infantile Deaths among Males for Certain Causes, Australia, 1911–1920.[1]

Month.	Premature Birth, Debility, Icterus and Sclerema.		Congenital Malformation.		Diarrhœa and Enteritis of Children Under Two Years of Age.	
	Observed.	Observed *minus* Calculated.	Observed.	Observed *minus* Calculated.	Observed.	Observed *minus* Expected.
January ..	1499	−47	240	−15	2365	+
February	1390	−21	216	−17	1962	+
March	1543	−3	204	−51	1804	+
April	1579	+83	232	−15	1513	+
May	1557	+11	272	+17	1120	−
June	1598	+102	274	+27	620	−
July	1688	+142	298	+43	454	−
August	1540	−6	295	+40	374	−
September ..	1475	−21	261	+14	423	−
October	1432	−114	239	−16	682	−
November ..	1425	−71	239	−8	1537	+
December	1489	−57	240	−15	2359	+
Total ..	18,215	−2	3,010	+4	15,213	

[1] The "expected" deaths have been calculated on 283 February days in 1911–1920, 310 January days *et cetera*. Data from the 1921 Census, "Intercensal Demography".

and indicates the different trends in the diseases for each of the first four weeks of life and for the first year.

The most recent experience in the incidence of various causes of death in the varying age periods is shown in Table VI, which is especially interesting. The results shown are percentages of total deaths from individual causes in each age group, for the ten years 1936-1945.

If the varying weekly and monthly group is analysed for separate causes, it is seen that under one week 95% of the deaths occur from causes directly following pre-natal or natal states; 2% are due to respiratory causes.

After the first week the emphasis alters quickly, and in the group "one week and under one month" only 73% of the deaths are due to pre-natal causes; but 12% are due to respiratory causes, and nearly 6% to diseases of the digestive tract.

In the group "one month and under three months" the diseases associated with pre-natal and natal causes are less important, although congenital malformations continue to account for 25%, diarrhœa and enteritis for 10%, and whooping cough for 7%; prematurity and congenital debility and other diseases peculiar to infancy, diseases of the digestive system and meningitis account for approximately 3% to 4% each.

The change of emphasis is still more noticeable in the later part of the year and swings right over to the highest rates of respiratory system diseases at 29%, of diarrhœa and enteritis at 19%, of whooping cough at 7%; and of meningitis and diseases of the digestive system each at 4%. In fact, 50% of the deaths are caused by respiratory diseases, diarrhœa and enteritis.

Comparative Mortality Rates in Nuptial and Ex-Nuptial Births.

Figure VI shows nuptial and ex-nuptial death rates among infants aged under one month and under one year, and is presented to indicate the comparative chances of survival of these infants.

The figures show that reduction in the ex-nuptial death rate in the first month is more marked than in that of the first year. One can assume that the reduction is in those deaths from concealment, neglect, exposure and other preventible causes. Figures show that the ratio of ex-nuptial to nuptial births has diminished, as well as the ratio to the total population.

There is, of course, evidence of great improvement in both, but the ex-nuptial rate shows a greater relative improvement than the nuptial rate. This would be due to a number of factors, both health and sociological. There has grown up in the community a respect for infant life hitherto unknown, on the grounds of the importance of racial and national survival, relating to the falling birth rate trends and the voluntary limitation of families. In addition, there is a consciousness that every child has a right to proper care and attention. This general change in attitude is reflected in the official recognition of *de facto* wives in certain instances.

Seasonal Influence.

Table VII demonstrates the expected seasonal influence in a number of diseases of the higher rate of death in the winter months from diseases such as prematurity, congenital debility and diseases peculiar to infancy including respiratory diseases; diarrhœa and enteritis show the reverse of high incidence in summer.

TABLE VI.[1]

New South Wales—Infantile Mortality.

Table Illustrating the Changing Emphasis on Various Causes as Age Group Changes. Experience in Ten Years 1936 to 1945.

Cause.	Percentage of Total Deaths in Each Age Group.			
	Under One Week.	One Week and Under One Month.	One Month and Under Three Months.	Three Months and Under Twelve Months.
Whooping cough	—	0·29	7·15	6·67
Diphtheria	—	0·17	0·29	1·16
Tuberculosis, all forms ..	—	0·04	0·17	1·23
Influenza	—	0·46	0·86	2·17
Measles	—	0·08	0·29	1·24
Meningitis (including epidemic cerebro-spinal meningitis)	0·07	1·00	3·63	4·63
Convulsions	0·25	0·62	0·75	0·70
Diseases of the ear and mastoid	0·01	0·04	0·23	0·65
Diseases of the respiratory system	1·55	12·37	24·32	29·31
Diarrhœa and enteritis ..	0·07	3·40	10·43	18·89
Diseases of the digestive system (excluding diarrhœa and enteritis) ..	0·19	1·37	3·05	4·00
Diseases of the genito-urinary system	0·04	0·91	1·04	1·05
Diseases of the skin ..	0·10	1·41	0·52	0·48
Congenital malformations	10·67	16·68	20·58	10·06
Congenital debility ..	4·80	8·13	4·15	2·02
Premature birth	50·29	27·85	7·09	0·46
Injury at birth	17·24	8·42	1·38	0·34
Other diseases peculiar to first year of life	12·97	11·12	3·46	0·57
All other causes	1·84	5·64	10·61	14·38
All causes	100·00	100·00	100·00	100·00

[1] The table has been prepared in such a way as to eliminate the repetition of any age group in the form of sub-totals; in this way a direct comparison can be achieved without clouding of the figures with repetition of earlier figures. (New South Wales Bureau of Statistics and Economics.)

Summary.

1. The improvement in the welfare of our infant population, with the age groups in which this improvement has occurred, is shown.

2. There has been little or no improvement in the most vulnerable age groups, particularly in the first week of life.

3. Improved pædiatric and obstetric practice, the dissemination of the knowledge of reasonable mothercraft practice to the majority of homes, the availability of reliable advice on mothercraft to all sections of the community, will have assisted in this improvement.

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PULMONARY TUBERCULOSIS IN THE MENTAL HOSPITALS OF WESTERN AUSTRALIA.

By FRANK PRENDERGAST and ALAN KING,
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X-RAY SURVEYS in the United States of America and in England within recent years have disclosed that a relatively high percentage of the inmates of mental hospitals have pulmonary tuberculosis. Incidences of 8·25% (Oechsli, 1949), 5·9% (Early, 1946), 7·2% (Ruskin, 1945), 8·8% (Leonidoff, 1943), and 6·2% (Altshuler and Bailey, 1941) have been disclosed.

A survey undertaken by the Tuberculosis Control Branch of the Western Australian Public Health Department in the institutions of the Western Australian Mental Hospitals Department has shown a similar prevalence, although possibly less active disease is present.

An initial survey was made on 35-millimetre film. All subjects with suspected tuberculosis were then radiologically examined on 17-inch by 14-inch films (November, 1948).

To obviate as far as possible any error in judgement in interpretation of films, serial X-ray pictures were taken in all presumptive and suspected cases six months later (May, 1949).

An attempt was then made to classify patients as suffering from active or inactive or suspected tuberculosis.

It is not the usual procedure of our Tuberculosis Control Branch to diagnose active pulmonary tuberculosis without laboratory confirmation, even if the X-ray appearance is characteristic. However, it was considered reasonable in a survey of this nature, especially as a definite percentage (40%) of subjects were considered to present evidence of cavitation.

Sputum confirmation has so far been possible in only 11% of cases diagnosed as of active disease, but difficulty in management of patients and shortage of facilities and staff have prevented full bacteriological confirmation.

It is possible that some of those patients classified as having inactive fibro-calcified lesions may yield positive findings from gastric lavage.

A total of 1445 patients, 849 male and 596 female, were examined at the four hospitals—Claremont Mental Hospital, Heathcote Reception Home, Lemnos Hospital and Greenplace Hospital. The results are shown in Table I.

A pleasing feature of the concomitant survey of the staff has been the low incidence disclosed amongst present members—0·3%. This is no doubt due to the fact that the awareness of the possibility of infection led to physical examination and X-ray check in past years (when some cases were found).

An analysis of the mental disorders among the 85 patients diagnosed as tuberculous gave the figures shown in Table II.

Various investigations have been made overseas into the problem of whether any particular type of mental disorder is more prone to be associated with tuberculosis. In particular this question has been investigated with regard

TABLE I.

Tuberculosis Status.	Subjects.		Total.	Percentage.
	Male.	Female.		
Active pulmonary tuberculosis ..	36	17	53	3·7
Inactive pulmonary tuberculosis ..	20	12	32	2·2
Suspected pulmonary tuberculosis (May, 1949)	7	11	18	1·2

to the contention that schizophrenics are predisposed to tuberculosis (Oechsli, 1949). However, different investigators have arrived at different conclusions on this question, and it does not seem that a predisposition to tuberculosis has as yet been clearly demonstrated (Early, 1946).

It has been suggested that any reciprocal relation between schizophrenia and tuberculosis is not simple and regular, and that it varies widely from case to case. This variation may depend not on a constitutional factor or on the schizophrenic's mode of life, but on constitutional and environmental factors acting together in a manner not yet altogether clear (Hurchsler and Perrier, 1946).

Schizophrenics, of course, form easily the largest group among the patients of the average mental hospital, generally over 50% (Leonidoff, 1943; Altshuler and Bailey,

TABLE II.

Mental Disorder.	Tuberculosis in Males.		Tuberculosis in Females.		Total.
	Active.	Inactive.	Active.	Inactive.	
Schizophrenia	17	13	7	4	41
Paraphrenia	5	3	4	2	14
Manic-depressive insanity	3	1	3	3	10
Senile dementia	5	1	1	1	8
Congenital mental deficiency	3	1	1	—	5
General paralysis of the insane	2	1	—	—	3
Involutional melancholia ..	1	—	1	1	3
Epilepsy	—	—	—	1	1

1941), and this must be remembered when one is considering the preponderance of schizophrenics among patients diagnosed as tuberculous.

A point of interest that was noticed in the present survey was the low incidence of infection diagnosed among patients with idiopathic epilepsy. Altogether 60 epileptics (32 females, 28 males) were radiologically examined, and of these one female was diagnosed as suffering from inactive tuberculosis. The incidence of significant lesions among the epileptics was therefore 1·6% as compared with 5·9% among the patients generally. No reference to a similar observation in other surveys has been noted; but in view of the claims made by some workers for an affinity between schizophrenia and tuberculosis and an antagonism between schizophrenia and epilepsy, the point is recorded as a matter of interest.

A correlation between the percentage of tuberculosis found and the length of stay in the institutions is indicated by the fact that 90% of the tuberculous patients have been inmates for more than five years. This is the usual finding, it being considered that the majority of patients contract their disease after admission to hospital (Oechsli, 1949; Lambiotte, 1949). In this relation, it is considered by Anderson (1949) that B.C.G. vaccination should be considered for all "Mantoux-negative" inmates, although the percentage of the latter is low.

Treatment in the cases we have found has been so far limited to increasing the amount of rest and to ensuring an adequate dietary.

It is interesting to find increasing enthusiasm abroad for active treatment of tuberculous patients in mental hospitals—"the problems in the management of psychotic patients with pulmonary tuberculosis are not insurmountable" (Lambiotte, 1949).

Artificial pneumothorax and thoracoplasty are considered quite practicable forms of treatment by some authors (Altshuler and Bailey, 1941; Lambiotte, 1949). One author (Leonidoff, 1943) considers artificial pneumothorax undesirable, but favours thoracoplasty.

One successful upper lobectomy for pulmonary tuberculosis in a patient with schizophrenia has been performed in Perth. As the schizophrenia appears relieved and the tuberculosis controlled, it is considered that in special cases resection may be the treatment of choice.

The survey raised a number of serious administrative problems in view of the shortages of staff and accommodation. However, it has been possible to isolate the affected patients in wards in which general improvements have been made and ventilation has been bettered. The knowledge that potentially infectious tuberculous patients have been isolated has had a good psychological effect on the staff.

Conclusion.

The need for initial mass radiography and serial X-ray examinations of patients in all mental institutions is considered established. These make possible (i) segregation and treatment of patients, (ii) protection of members of the medical and nursing staffs, (iii) prevention of the return to the community of potentially infective patients with pulmonary tuberculosis, (iv) the usual follow-up examination and X-ray control of contacts and relatives of patients who are found to have tuberculosis.

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DIAGNOSTIC PROBLEMS IN RENAL FAILURE.[1]

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IN 1694 Dekker described a test for the detection of albumin in the urine. One hundred and thirty-three years later, Richard Bright applied this test in a series of cases of dropsy; he boiled the urines in a pewter spoon over a tallow candle, and added a few drops of vinegar. He found that those people who had albuminuria also had enlarged hearts and high blood pressure, and that in their kidneys at autopsy were found pathological changes, which could be divided into three groups: (i) a "state of degeneracy" of the kidneys; (ii) changes which now suggest glomerular nephritis; (iii) renal sclerosis.

In spite of the great accumulation of knowledge since then, do we know so much more about the clinical aspects of nephritis than Richard Bright did one hundred and twenty-two years ago? The physiologists seem to have outstripped the clinicians and the pathologists in this field.

In renal failure from any cause, the first problem is to recognize it, when it presents. This is not always so simple as it would appear to be. I know that I have overlooked it on more than one occasion, and several patients with renal failure have brought letters from their doctors with a variety of diagnostic suggestions far removed from the kidney.

The second problem, which may be very difficult, is to determine the cause of the renal failure.

FREQUENCY AND ÆTIOLOGY OF RENAL FAILURE.

Investigation of 440 consecutive post-mortem examinations at the Royal Hobart Hospital showed that death was due to renal failure in 49 (11·1%). Therefore we may expect about one patient in every ten to die from some form of renal failure.

The 49 cases may be divided into four groups: (i) glomerular nephritis—14 cases (one acute, one subacute, 12 chronic); (ii) renal arteriosclerosis—14 cases; (iii) obstruction to the urinary tract—13 cases (10 prostatic, two carcinomatous, one congenital); (iv) miscellaneous group—eight cases. In the last group there were two cases of polycystic disease, and there was one case each of renal tuberculosis, amyloid disease, renal calculi, pyelonephritis, gout and tuberous sclerosis.

Apart from these 49 renal deaths, examination of 76 subjects dead from other causes revealed pronounced renal arteriosclerosis, and 45 of them had died from vascular lesions.

Acute Renal Failure.

I wish first to discuss briefly acute renal failure.

The main clinical conditions associated with it are set out in the following list (modified from Joekes, 1949): (i) obstruction to the renal tract—by prostatic enlargement, calculi, carcinoma, stricture *et cetera*; (ii) acute glomerular nephritis; (iii) hepato-renal failure; (iv) cortical necrosis; (v) lower nephron nephrosis (renal anoxia syndrome).

A large number of different conditions may produce tubular degenerative changes characteristic of lower nephron nephrosis, the most important being as follows: (*a*) due to sulphonamides—either crystal blocking or sensitivity; (*b*) pigment nephrosis—from hæmolytic infections, transfusion reactions, crush syndrome, severe burns *et cetera*; (*c*) "shock" kidney—following surgical operations, severe intraabdominal hæmorrhage, prolonged vomiting or diarrhœa *et cetera* ("extrarenal azotæmia"); (*d*) due to chemical poisons—mercury, carbon tetrachloride, gold salts, arsenic.

The pregnancy anurias form the largest clinical group.

In septic abortion the lesion is usually pigment nephrosis, due to hæmolytic infection. In non-septic abortion, either

[1] Read at a meeting of the Tasmanian Fellows and Members of The Royal Australasian College of Physicians, Launceston, September 24, 1949.

pigment nephrosis or bilateral cortical necrosis may occur. Severe post-partum hæmorrhage may produce prerenal azotæmia, and cortical necrosis is usually found in fatal cases of ante-partum hæmorrhage.

Clinical Picture of Acute Renal Failure.

Whatever the cause of acute renal failure, the clinical features are much the same; but at the outset the symptoms may be completely masked by the gravity of the causal factor—for example, hæmorrhage, shock or infection—and the anuria may escape notice.

With the initial shock there is a steep fall in the blood pressure, resulting in anuria or oliguria. There will be a complaint of weakness, nausea, vomiting, and pains in the loins or abdomen. If the patient survives the shock, the blood pressure begins to rise again, and nitrogen retention steadily increases. Acidosis may be evident; but neither this nor the degree of nitrogen retention bears any relation to prognosis. Even when anuria persists for two or even three weeks, the condition is still reversible (because of regeneration of tubular epithelium) provided fluids are not given in excess. Many patients have died of pulmonary œdema as the result of "forcing fluids" (Strauss, 1948; Darmady, 1949).

If a patient can be kept alive for two or three weeks, natural diuresis will occur and he will recover completely, although the blood urea level will characteristically remain high for weeks or even months before returning to normal.

In the diagnosis of acute renal failure, renal function tests are, of course, useless, as there is practically no renal function, so that the history and the chemical examination of the blood are all-important.

Although this condition is not so common as chronic renal failure, there have been examples of the types listed, with the exception of the chemical poisons, in the Royal Hobart Hospital in the last two years. The following brief notes illustrate some of the points mentioned.

CASE I.—A woman, aged thirty-one years, two months pregnant, had had instruments used on her the day before her admission to hospital. She complained of lower abdominal pain and vaginal bleeding. She showed signs of shock with low blood pressure, and considerable tenderness was present in the lower part of the abdomen and the fornices. *Bacillus welchii* was cultivated from the cervix. She was given intensive treatment with penicillin, gas-gangrene antiserum and blood transfusions; but oliguria developed, and for five days she passed not more than one ounce of dark urine each twenty-four hours. She became drowsy, and the blood urea level on the sixth day rose to 240 milligrammes per 100 millilitres. She died on the ninth day. At autopsy, both kidneys were enlarged, and the substance was dark on section. The cortex appeared normal. On microscopic examination the glomeruli looked normal, but the tubular epithelium was grossly degenerated, and the tubules contained pigment casts. The diagnosis was pigment nephrosis.

CASE II.—A married woman, aged fifty-one years, developed shivering, anorexia, and slight delirium, and her temperature rose to 103° F. On the fourth day jaundice was evident, and she became very ill, her appearance suggesting acute yellow atrophy of the liver. She had a dry brown tongue, and was sleepy and disorientated; she vomited and became anuric. The blood urea content was estimated at 110 milligrammes per 100 millilitres. The urine contained albumin, bile pigments, a moderate number of granular casts and a few red blood cells. Her blood pressure was 100 millimetres of mercury (systolic) and 70 millimetres (diastolic). Her fluid intake was limited to about 1500 millilitres per day. On the fourth day of the anuria, diuresis occurred, and a week later her condition was greatly improved, and the jaundice had almost disappeared. Her blood pressure rose to 220 millimetres of mercury (systolic) and 120 millimetres (diastolic), which was the same as it had been for the previous two years. She made a good recovery, and her renal and hepatic functions appear now to be normal. The diagnosis was thought to be essential hypertension and infective hepatitis with hepato-renal failure.

Little seems to be known of the mechanism of hepato-renal failure.

Chronic Renal Failure.

The most important causes of chronic renal failure are as follows: (i) obstruction to the urinary tract; (ii) essential or malignant hypertension with secondary nephritis; (iii) glomerular nephritis; (iv) renal arteriosclerosis; (v) renal infections, especially chronic pyelonephritis and tuberculosis; (vi) developmental anomalies of the kidneys, especially polycystic disease; (vii) septicæmia—usually due to *Streptococcus viridans*—with subacute bacterial endocarditis and secondary glomerular nephritis; (viii) nephrosis; (ix) diseases of the renal arteries—disseminated *lupus erythematosus* and *periarteritis nodosa*; (x) renal disease secondary to multiple myeloma, leuchæmia and Hodgkin's disease.

Clinical Picture of Chronic Renal Failure.

The full-blown picture of uræmia is well known. The patient is semi-conscious, and has a yellowish pallor and ammoniacal breath, which he puffs in and out with a hissing sound. He twitches, hiccups and vomits, and may show signs of hypertensive failure. Such a patient is sometimes sent to hospital because of mental disturbance and mania.

However, it is the insidious type of chronic renal failure that I wish to discuss, because it is less obvious, yet is not uncommonly met. The patient gives a vague history of ill health without localizing symptoms, or only those that introduce the "red herring". There were several cases of this type among the 49 that ended fatally, and looking through the clinical notes, I find that loss of weight and breathlessness were most often mentioned. Some of these people showed no evidence of cardiac damage, so that the breathlessness was probably due to the anæmia of renal failure.

Weakness, mild dyspeptic symptoms, an occasional attack of unexplained vomiting or diarrhœa, and complaint of headache were fairly common symptoms. A previous history of kidney trouble was frequently denied.

Examination nearly always confirmed weight loss. Anæmia of some degree was the rule. Hypertension and cardiac complications were often found, but by no means always, and sometimes the examination gave entirely negative findings.

Chronic Glomerular Nephritis.

It is well known that albuminuria tends to diminish in the terminal stages of chronic nephritis, but it was only recently that I appreciated the fact that albumin may be absent from the urine for days at a time; yet this was pointed out in 1845 by Rees.

In the urine of a patient recently under observation because of chronic nitrogen retention, a search for albumin was made on twenty-eight consecutive days before it was found, and in a proved case of chronic nephritis the first fourteen tests failed to reveal albumin. Fourteen of the forty-nine patients previously mentioned died without having had a positive result to tests for albuminuria. However, it is possible that frequent tests were not made in these cases, because on clinical grounds some deaths were thought to have been due to uncomplicated congestive cardiac failure.

Another fact to be kept in mind in these people with insidious renal failure is that their blood pressures may be normal, and there may be no evidence of previous hypertension, such as cardio-vascular hypertrophy or retinal changes. The following case illustrates these points.

CASE III.—A single woman, aged thirty-seven years, was sent to hospital with a diagnosis of nervous dyspepsia. She had been in good health until six months previously, when she began to feel generally "below par" with vague indigestion, occasional vomiting for no apparent reason, and a steady loss of weight with weakness. She was breathless on effort. She denied any present or past urinary symptoms. She was thin and pale. Her heart was normal, her blood pressure was 110 millimetres of mercury (systolic) and 60 millimetres (diastolic), her fundi were normal, and no albumin was found in her urine. For the first week in hospital she seemed fairly well and complained only of weakness. During the second week she vomited occasionally, and diarrhœa developed. In the third week she became drowsy and her blood pressure dropped to 70 millimetres of mercury (systolic) and 30 millimetres (diastolic). Anuria

occurred, and she presented a typical uræmic picture before she died on the twenty-sixth day in hospital.

A number of investigations were carried out.

A trace of albumin was found in the urine after seven days, and again a week later. A few granular casts were seen on several occasions, but no cells. The volume of urine varied, but was usually about 30 ounces per day. The specific gravity was never above 1·012. The chloride content was 2·3 grammes per litre during the second week, and during a urea concentration test, all specimens of urine contained less than 1% of urea.

Blood examination revealed mild normocytic anæmia. The urea content of the blood on the patient's admission to hospital was 76 milligrammes per 100 millilitres, rising to 264 milligrammes per 100 millilitres at the end of the third week. The plasma chloride content (as sodium chloride) was 640 milligrammes per 100 millilitres. The total serum protein content was 5·3 milligrammes *per centum*. The carbon dioxide combining power was 36·6 volumes *per centum*. The fasting glucose content was 149 milligrammes per 100 millilitres. Excretion pyelography revealed no excretion after thirty minutes. The cerebro-spinal fluid was normal.

At the post-mortem examination fibrinous pericarditis was found. The heart and coronary arteries were normal. No other abnormalities were found except in the kidneys. There was no renal obstruction. The kidneys were normal in size. Under the capsule, the surface was finely granular. The substance of the kidney was fibrous and the cortex pale. On microscopic examination, Bowman's capsule was found to be thickened, but the tufts looked fairly normal. Some casts were present in the tubules, and the tubular epithelium was grossly degenerated.

The features of this case that I wish to emphasize are: the absence of any history of infection, previous kidney trouble or hypertension; the long periods during which there was no albuminuria; the absence of œdema, cardio-vascular involvement and retinitis; and the somewhat sparse post-mortem findings.

A case now under observation closely resembles this case in every detail.

Renal Arteriosclerosis.

Fourteen of the forty-nine renal deaths were thought to have been due to renal arteriosclerosis. The kidneys were granular, and pronounced changes in the arterioles were noted. Although these people were aged, their average age being seventy-nine years, the renal lesions were not typical of the senile kidneys which Clifford Allbut described as "the starved but not corrupt kidneys sufficient for the smaller life of the elderly". No doubt hypertension was an important ætiological factor, and sometimes partial prostatic obstruction and renal infection.

Elderly people are so prone to frequency of micturition that this symptom is apt to be ignored, and their feebleness and cerebral symptoms are put down to senility and cerebral arteriosclerosis.

Many of these cases were complicated by congestive cardiac failure and œdema, and it is in this type of illness that a sudden collapse occurs on rare occasions—a form of lower nephron nephrosis produced by salt depletion and dehydration from the use of mercurial diuretics. The clinical notes of at least one patient suggested that this had occurred.

Chronic Pyelonephritis.

The clinical features of acute and recurrent pyelonephritis, and the surgical complications, whether bilateral or unilateral, are usually fairly clear cut; but the chronic, latent or "burnt out" stage of the disease may be difficult to recognize. There should be a previous history of urinary symptoms, but this may have been forgotten. The clinical picture is one of prolonged and insidious renal failure, mild in degree. Even after nitrogen retention is well established, death may not occur for years, and there may be no renal symptoms. Anæmia develops slowly, and hypertension is found in about half the cases.

Pyuria is constant, though often slight, and this may be the only factor that distinguishes the disease from the terminal stage of chronic nephritis. Albumin is usually constantly found in the urine, and although it is often only in small quantities, yet the amount is in excess of that to be expected from the number of pus cells present. Red cells may also be found in the deposit, and also bacteria, but these are usually transient. Casts are absent as a rule.

Polycystic Disease of the Kidneys.

People with polycystic kidneys may live to a ripe age and die of other diseases without ever having suffered any renal disorder. Surgeons may encounter the disease because of hæmaturia, pyelonephrosis, perinephritic abscess or renal calculi. The condition has been reported by obstetricians, the baby's huge kidneys having delayed labour. Physicians may be consulted because of hypertension, congestive cardiac failure or chronic renal failure.

There have been three instances of polycystic disease at the Royal Hobart Hospital recently, and all three patients have died from uræmia. All cases were complicated by gross infection of the renal tract, and in one calculi were present. The main difficulty in diagnosis is due to the fact that the tension in the cysts may be low, so that the kidneys, although much enlarged, cannot be felt (Addis, 1948). Excretion pyelography is seldom of any help, because by the time the disease produces symptoms, renal function is so poor that there is insufficient concentration of the dye, and retrograde pyelography is unsafe in the presence of sepsis.

In the stage of chronic renal failure, the clinical features once again simulate the terminal stage of nephritis.

The use of "Diodrast" is said to assist the diagnosis.

Table I illustrates the similarity of the findings in the three diseases discussed.

TABLE I.

Finding.	Findings in the Terminal Stage.		
	Glomerular Nephritis.[1]	Chronic Pyelo-nephritis.[1]	Polycystic Disease.[1]
Hypertension ..	Usually present.	In about half.	Less common.
Arteriosclerosis ..	Usually present.	Sometimes present.	Usually present.
Anæmia	Always present.	Usually present.	Not present as a rule.
Nitrogen retention	Always present.	Always present.	Always present.
Albuminuria ..	Usually present.	Usually present.	Usually present.
Casts	Always present.	Absent.	Sometimes present.
Cells	Epithelial cells present.	Pus cells a constant finding.	Pus cells often present.
Red blood corpuscles	A few present.	A few present.	Number may be gross.
Bacteria	Absent.	Intermittently present.	Often present.

[1] Symptoms of chronic renal failure.

Summary.

The common causes and clinical features of acute renal failure are briefly discussed, with short case notes illustrating pigment nephrosis and hepato-renal failure.

Some of the important causes of chronic renal failure are reviewed.

Forty-nine (11·1%) of 440 subjects of autopsies at the Royal Hobart Hospital had died of renal failure. Fourteen of these died from Bright's disease, fourteen from renal arteriosclerosis, thirteen from urinary obstruction, and eight from other renal lesions.

The clinical picture of the insidious form of chronic renal failure is described, with an illustrative case history.

Emphasis is laid on the fact that albumin may be absent from the urine for days or even weeks at a time in chronic renal failure, and examination of the cardio-vascular system may show no abnormality.

The similarity of the clinical features in the terminal stages of glomerular nephritis, chronic pyelonephritis and polycystic kidneys is discussed.

Acknowledgement.

Dr. R. Pitney kindly supplied the post-mortem details and rendered much assistance with the clinical histories.

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Reports of Cases.

SNAKE BITE BY A TAIPAN WITH RECOVERY.

By C. C. REID AND H. FLECKER,
Cairns, North Queensland.

THE only cases so far recorded in the medical literature of snake bite by the taipan, *Oxyuranus scutellatus*, are three reports in THE MEDICAL JOURNAL OF AUSTRALIA in 1940, and three more in the same journal in 1944, by one of us (H.F.). Of these patients only one recovered—the only instance known so far. Unfortunately the snake was buried before expert examination of it could be made, but another snake which was produced was stated to be a duplicate and was shown to be the taipan.

The present account deals with the first report of a case of snake bite by the taipan with recovery in which the snake has been secured and identified, the determination having been made by Professor Donald Thomson of the University of Melbourne.

Clinical Record.

The victim was G.R., aged nineteen years, an Australian aboriginal boy of excellent physique, stationed at the Lutheran Mission, thirty miles from Cooktown, who was bitten at 2.30 p.m. on August 16, 1949; he was stacking timber when he felt a bite on the outer aspect of the right ankle. Although he did not see the snake himself, it was seen and killed by his companions, the head being afterwards forwarded for examination. The bite was through his boot, thick sock and very thick skin, and as this reptile is provided with a pair of very long curved fangs it is able to do this. Probably no other Australian snake with much smaller fangs could accomplish this feat. A tourniquet was immediately applied around the right thigh and the site of the puncture incised with a chisel. Apparently a vein was cut at the ankle and considerable hæmorrhage occurred. The patient does not know what was used as a tourniquet.

It appears that the patient became ill about fifteen to thirty minutes after having been bitten. The first thing he noticed was that he could not open his eyes. This condition was rapidly followed by nausea (he does not remember whether he vomited) and difficulty in breathing, and he felt that he was choking. Extreme drowsiness was present. He did not remember anything more until he reached the Cairns Base Hospital at 10 a.m. on August 17.

The victim was taken to Cooktown Hospital, where there was no regular medical attendant; but Dr. A. C. Thomas, of Bentleigh, Victoria, who was at Cooktown as a visiting tourist, was urgently sent for. Dr. Thomas supplied the following notes.

> At 6.30 p.m. I saw him. He was almost unconscious, quite pulseless and cold with dry skin. Counted by auscultation, his heart beat was about 140 per minute and rather irregular. Occasionally he tossed himself in the bed. There was only one hot water bag available and no antivenin. As the boy appeared to be moribund it seemed that no treatment had much prospect of being successful. However, it was decided to try intravenous glucose and saline and to see if it could be possible to get some antivenin from Cairns.
>
> A supply of antivenin arrived at 9.30 p.m. by aerial ambulance. The same antivenin (for tiger snake) is used for all snake bites and 9,000 units are advised by the intravenous route for taipan bite. I gave him this dose by injecting it through the rubber tube into the glucose solution that was already being given. Within a very few minutes the youth was pulseless again, but not for more than about a quarter of an hour. I gave him 3,000 units at about 11.30 p.m. and again at about 2.30 a.m., 17.8.'49. His pulse rate was still 130 at 4 a.m. and during the night had not been palpable on many occasions. At about midnight I passed a catheter and withdrew over two quarts of urine. At about 3 a.m. the boy was more or less conscious. He complained of double vision, being unable to open his eyes fully, and weakness in his arms. By 4 a.m. he had had seven "Soluvacs" of varying types of solution of saline and glucose—I simply had to use what was available (it was all very old stock), and I started him on his eighth flask before I left the hospital to catch my launch. He was to be flown to Cairns at about 8 or 9 a.m.

The patient's condition upon his arrival at the Cairns Base Hospital was fair; he was drowsy and restless, his respirations numbered 20 per minute, and he was distressed and rather "moist". His pulse rate was 120 per minute, the pulse being of small amplitude and regular. His blood pressure was 130 millimetres of mercury, systolic, and 70 millimetres, diastolic. His temperature was 97·8° F. He was cold and clammy and unable to open his eyes, holding his upper eyelids open with his clammy fingers. He complained that his "jaw was locked" and that he was unable to open his mouth widely although it could be opened passively. He appeared to have some facial weakness with difficulty in swallowing, in coughing, and in the protrusion of his tongue, which was in the middle line.

No ocular muscular paralysis was detected.

The pupils reacted to light. All the tendon reflexes were extremely sluggish, and the plantar reflexes were absent.

The senses of smell and taste were not tested upon his arrival.

There appeared to be considerable weakness of the arms and some weakness of the legs. He was able to reply to questions, but was very drowsy.

Immediately on his arrival 6000 units of antivenin were given intramuscularly with six minims of adrenaline hydrochloride hypodermically, and the patient was given oxygen continuously by nose. Shortly afterwards these measures were followed by the intravenous administration of 25% dextrose solution (with one millilitre of "Neosynephrin" per litre) through a cannula inserted in a vein over the right ankle. To maintain the alkalinity of the urine, *Mistura Potassii Citratis* (two drachms) was given every two hours for six doses, then every four hours.

Shortly afterwards the blood pressure rose to 160 millimetres of mercury, systolic, and 60 millimetres, diastolic, and the pulse was 140 per minute, the pulse being of better volume. At noon, 20 ounces of urine were passed; the urine was alkaline, and contained a trace of sugar, but no albumin or blood. Intranasal oxygen therapy was suspended during the day and the patient's general condition remained fair.

On August 18 his condition was slightly improved; the muscular weakness was slightly less. His olfactory sense was disturbed—things smelt and tasted "different" and usually unpleasant. The intravenous administration of 5% dextrose solution was stopped after a total of two and a half litres had been given following an initial litre of 25% solution.

On August 19 his condition was much the same. Antitetanic serum (1000 units) was given in view of the chisel wound at the ankle, and some local reaction occurred. Fifty milligrammes of "Benadryl" were given three times a day.

On August 20 his condition was much improved. He could open his eyes fairly well, though slight facial weakness was still present. The senses of taste and smell were normal. He had slight weakness in the arms, more pro-

nounced on the right. There was no weakness in the legs. He had no difficulty in swallowing and his reflexes were normal. His blood pressure was 140 millimetres of mercury, systolic, and 70 millimetres, diastolic.

On August 21 there was a further decrease in the muscular weakness.

On August 22 the patient's condition was unchanged except for a headache.

On August 23 malaise, headache, some swelling of the eyelids and hands, and the presence of pruritus revealed serum sickness. Ten millilitres of calcium gluconate solution were given intramuscularly per day, 50 milligrammes of "Benadryl" were given every six hours, eight minims of *Liquor Adrenalinæ Hydrochloridi* were given every six hours, and *Lotio Calaminæ cum Phenol* was prescribed. His hæmoglobin value was 40%, and the red blood cells numbered 1,720,000 per cubic millimetre. Two "Polyhæmen" tablets were given three times a day.

On August 24 the patient was more comfortable and his skin was less itchy. On August 25 he was comfortable. Treatment with adrenaline and calcium gluconate was suspended. On August 26 he was well. Slight facial weakness and slight weakness in the right arm were still present; otherwise his condition was normal. On August 27 he was well. Slight epistaxis had occurred during the night, but had not required treatment. His blood pressure was 130 millimetres of mercury, systolic, and 70 millimetres, diastolic.

On August 28 he was well, and out of bed. Apart from weakness, no abnormality was detected. His hæmoglobin value was 43%. On August 29 he was up and about.

On September 1 examination of his urine revealed a faint trace of albumin, with an occasional leucocyte, but no casts, red cells or organisms were detected.

On September 3 the hæmoglobin value was 50%, the red blood cells numbered 3,240,000 per cubic millimetre and the leucocytes 10,800 per cubic millimetre.

On September 4, the nineteenth day of illness, the patient was discharged from hospital. He had no residual symptoms at all—no paresis or disorders of sensation, no mental symptoms. Some anæmia was present. His blood pressure was 130 millimetres of mercury, systolic, and 70 millimetres, diastolic, and his temperature was normal. He was advised to continue taking "Polyhæmen" tablets and to report to Cooktown Hospital from time to time.

The severe anæmia was thought to be due to loss of blood when the ankle was incised. Hæmolysis was not a pronounced feature. The patient was never clinically jaundiced. Albuminuria occurred only occasionally and was never more than slight. The specific gravity of the urine varied between 1012 and 1020. The temperature rose to 102° F. shortly after administration of the first litre of 25% dextrose solution was begun. There was slight elevation of temperature (99·5° to 100° F.) daily until the day before the patient's discharge from hospital. Finally, recovery from the effects of the snake bite itself appears to be complete, the residual anæmia being attributed to the first-aid measures.

Comment.

The following factors may each be considered important in contributing to the recovery of the patient: (i) the wearing of thick boots and socks and possession of a thick skin by the victim (no doubt these all limited the amount of poison injected by this most dangerous of all Australian snakes); (ii) the rough incisions with a chisel, made as a first-aid measure with much bleeding, perhaps removing much of the venom injected; (iii) the administration of glucose and saline solution by Dr. A. C. Thomas at Cooktown four hours after the injury; (iv) the administration of antivenin (tiger snake) seven hours after the injury.

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Reviews.

AN ATLAS OF POSITIONS FOR RADIOGRAPHY.

A TWO-VOLUME WORK entitled "Atlas of Roentgenographic Positions" by Vinita Merrill (Educational Director of the Picker X-ray Corporation) has been received.[1]

The publication deals with all the usual standard positions adopted for radiography, and many unusual positions for displaying various structures. It is an elaborate work with many hundreds of fine photographs with concise individual descriptions. Each region of the body is dealt with in order, and an excellent description of the anatomical structures with drawings of the part under examination precedes the X-ray text. A bibliography is appended to each volume and the full index is given at the end of each volume, which is of great convenience in a multi-volumed work. A glossary of medical terms is included at the end of Volume I.

In the early part of the work the author refers to various points which the technician must appreciate to make the ideal radiograph of the part under examination, for example, relationship of structural shadows, detail of various tissues, effects of different focal spots, distortion or magnification of the images, effects of slight motion, poor screen contact *et cetera*. It is recommended that a short history be available to allow the technician to prepare a film with the maximum information possible for the radiologist to interpret. The technician is urged to see that the radiographic rooms are kept constantly clean, and that good ventilation is available (air should be changed three times an hour), and patients should not be exposed to draughts. Technicians should be taught strict asepsis, especially where any surgical procedures are carried out in the department.

When preparation before radiography is necessary, all instructions should be explained carefully to the patient, and when complicated, should be handed to the patient in writing. The author also stresses the care that must be exercised in the handling of patients, so as to cause as little inconvenience and disturbance as possible. The technician must also be careful to mark all radiographs carefully for identification.

A useful chart is presented in the section on general anatomy, which shows the normal development of important bones and epiphyses. The anatomical description of each region is adequate and clear, and the line drawings are excellent. In the illustrations of the various positions, the part being examined is shown resting on the film on the table together with the tube and direction of the X rays, and the resultant radiograph is reproduced below the letterpress and photographs.

In addition to the usual skeletal structures there are sections devoted to the examination of the body cavities, soft structures and digestive and excretory systems, and the female reproductive organs. The work is a most important one for both radiologist and technician and shows a distinct advance on any previous publication.

It is a pity that the price is so high, as it puts the work beyond the means of many technicians.

Notes on Books, Current Journals and New Appliances.

A GUIDE TO PACIFIC ISLAND DIETARIES.

IN "A Guide to Pacific Island Dietaries", J. C. R. Buchanan has addressed himself primarily to the students of the Central Medical School in Suva, but he expresses the hope that the book will also be of assistance to "health sisters, nurses and others—perhaps even doctors themselves—whose duty it is to advise on the diets of individuals and communities or to frame diet scales for schools and other institutions".[2] The first chapter records briefly and simply

[1] "Atlas of Roentgenographic Positions", by Vinita Merrill; Volumes I and II; 1949. St. Louis: The C. V. Mosby Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 12" × 8½"; Volume I, pp. 320, Volume II, pp. 400, with many illustrations, some coloured. Price: £11 5s. for the two volumes.

[2] "A Guide to Pacific Island Dietaries", compiled by J. C. R. Buchanan, M.D., F.R.C.P.(E.), D.T.M. and H., on behalf of the South Pacific Board of Health; 1947. 7¼" × 4¼", pp. 84.

general dietetic principles as they are at present accepted. The second chapter collates current information on the nature and composition of certain foodstuffs in daily use in the islands of the tropical South-West Pacific. The remaining chapters deal with dietary allowances and dietary scales for various classes of people, according to age, sex, occupation *et cetera*, special attention being paid to the infant and the pre-school child, and the book concludes with tables of food values of tropical foods. The author makes no claim to completeness or finality in this compilation and hopes to stimulate readers to add to the sum of knowledge from their own observations and to demand classification and analyses of many of the foodstuffs which have so far not been completely examined. The book should be of use to all who are concerned with tropical food and diets.

MEDICAL PROGRESS.

IN "Medical Progress, 1949",[1] the editor-in-chief, Lord Horder, has brought together a group of leaders in their respective fields of medicine and surgery to survey their own subjects: Professor Robert Platt writes on medicine, Sir Charles Max Page on surgery, Professor F. J. Browne on obstetrics and gynæcology, T. Jenner Hoskin on cardiology, William Gunn on acute infectious diseases, Sir Leonard Parsons on pædiatrics, H. V. Morlock on pulmonary tuberculosis, Sir Harold Scott on tropical medicine, E. L. Middleton on industrial medicine, Walter Montague Levitt on medical legislation, Professor J. R. Marrack on chemical pathology, and Professor W. J. Dilling on pharmacology and therapeutics. Each presents a brief critical review of recent advances, Professor Dilling's article including a commentary on the British Pharmacopœia, 1948. These articles make up Part I of the volume. Part II provides details of articles and preparations added to the British Pharmacopœia with an outline of the clinical application of each. Part III consists of abstracts from current literature. This third part would be much improved by division of each page into two columns; the present lay-out, in which closely packed lines of small type extend across the full width of a broad page, makes reading irritating and comprehension difficult. The abstracts, which are informative and comprehensive, are nearly all dated 1947 (the introduction to the volume is dated January, 1949), though this is not necessarily a disadvantage from the point of view of care and perspective in selection. The volume fulfils more than adequately its purpose of keeping up to date "The British Encyclopædia of Medical Practice".

INCOMPATIBILITY IN PRESCRIPTIONS.

FOR the assistance of the general practitioner who has not been sufficiently instructed as a student in the principles of prescribing, Thomas Stephenson prepared in 1915 the first edition of "Incompatibility in Prescriptions and How to Avoid It".[2] A gap of fourteen years has occurred since the last edition in 1935; during that time the original author has died and the latest edition has been prepared by James Burnet. Although less obviously important now, when simplicity in prescribing is the order of the day, it nevertheless contains much useful information and will probably provide some surprises for many who write prescriptions with confidence. It is in two parts. In Part I, incompatibility is discussed in three groups—chemical, physical or pharmaceutical and therapeutic. Part II is "a dictionary of incompatibilities"; in it the examples of incompatibility already discussed in Part I are summarized under the names of the drugs (arranged alphabetically by their English names), other useful data, such as doses, solubility *et cetera*, being added. An index adds further to the ease of reference in a useful little book.

[1] "The British Encyclopædia of Medical Practice Including Medicine, Surgery, Obstetrics, Gynæcology and Other Special Subjects: Medical Progress, 1949"; Editor-in-Chief, the Right Honourable Lord Horder, G.C.V.O., M.D., B.Sc., F.R.C.P.; 1949. London, Africa, Australia, Canada and New Zealand: Butterworth and Company (Publishers), Limited. 9½" × 6½", pp. 468. Price: 44s.

[2] "Incompatibility in Prescriptions and How to Avoid it with a Dictionary of Incompatibilities", by Thomas Stephenson, D.Sc., Ph.C., F.R.S.E.; revised by James Burnet, M.A., LL.B. (London), M.D., F.R.C.P.E.; Fifth Edition; 1949. Edinburgh: "The Prescriber" Publishers, Limited. 8" × 5½", pp. 76. Price: 10s.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Communism, Capitalism and Christianity", by Emil Brunner, Dr.Theol., and Norman P. Goldhawk, M.A.; 1949. Sydney: Angus and Robertson. 7" × 4⅜", pp. 48.

The author discusses: (*a*) "The Christian Witness to the Order of Society and National Life." (*b*) "The Church's 'No' to Communism." (*c*) "The Church's 'No' to Capitalism."

"Mongolism (Peristatic Amentia)", by M. Engler, M.D.; 1949. Bristol: John Wright and Sons, Limited. London: Simpkin Marshall, Limited. 8½" × 5½", pp. 220, with few illustrations. Price: 21s.

Intended as a summary of the literature on mongolian idiocy and an attempt to explain its cause.

"Arterial Hypertension: Its Diagnosis and Treatment", by Irving H. Page, M.D., and Arthur Curtis Corcoran, M.D.; 1949. Chicago: The Year Book Publishers, Incorporated. 8" × 5¼", pp. 402, with 20 illustrations.

One of the "General Practice Manuals" of which twenty have been published.

"The Examination of Waters and Water Supplies (Thresh, Beale and Suckling)", by Edwin Windle Taylor, M.A., M.D., B.Ch. (Cantab.), M.R.C.S., L.R.C.P., D.P.H. (London); Sixth Edition; 1949. London: J. and A. Churchill, Limited. 9½" × 6", pp. 836, with 52 illustrations. Price: 70s.

Deals with the analysis and purification of waters for public supply.

"Isotopic Tracers and Nuclear Radiations: With Applications to Biology and Medicine", by William E. Siri; 1949. New York, Toronto and London: McGraw-Hill Book Company, Incorporated. 9" × 6", pp. 574, with 136 illustrations. Price: $12.50.

Intended to bridge the gap between books intended solely for the nuclear physicist and those describing the results of research in which radioactive isotopes and nuclear radiations were used.

"Doctors East Doctors West: An American Physician's Life in China", by Edward H. Hume, M.D.; 1949. London: George Allen and Unwin, Limited. 8½" × 5½", pp. 282, with illustrations. Price: 12s. 6d.

The story of the author who went to China to found the Yale-in-China Medical School.

"Operative Obstetrics: A Guide to the Difficulties and Complications of Obstetric Practice", by J. M. Munro Kerr, LL.D., F.R.C.O.G. (Hon. Causa), M.D., F.R.F.P.S.G., and J. Chassar Moir, M.A., M.D., F.R.C.S.E., F.R.C.O.G.; Fifth Edition; 1949. London: Baillière, Tindall and Cox. 9½" × 6½", pp. 950, with 390 illustrations. Price: £3 3s.

Intended for obstetric specialists or prospective specialists and for general practitioners who are seriously interested and actively engaged in obstetric practice.

"Golden Jubilee World Tribute to Dr. Sidney V. Haas: In Honor of His Pioneering Contribution to Celiac Therapy and the Treatment of the Hypertonic Infant, and of the Completion of His Fiftieth Year of Medical Practice"; 1949. New York: The Committee for the Golden Jubilee Tribute to Dr. Sidney V. Haas. 9" × 6", pp. 44, with illustrations.

A full account of the tribute that was paid in the spoken and written word to Dr. Sidney V. Haas at New York in April, 1949.

"An Introduction to Clinical Perimetry", by H. M. Traquair, M.D., F.R.C.S. (Edinburgh), with a foreword by Norman M. Dott, M.B., Ch.B., F.R.C.S. (Edinburgh); Sixth Edition revised and enlarged; 1949. London: Henry Kimpton. 10" × 7½", pp. 352, with 257 illustrations. Price: 42s.

An attempt to introduce the reader to the essential principles of perimetry considered in their clinical aspect, and not to deal exhaustively with the subject.

The Medical Journal of Australia

SATURDAY, JANUARY 21, 1950.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: surname of author, initials of author, year, full title of article, name of journal without abbreviation, volume, number of first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

ONCE UPON A TIME: UTOPIA MEDICA.

ONCE upon a time, in the not so very long ago, geographers and sociologists are said to have recorded the existence of a populous and highly civilized community which lived in a country called by the unusual name of "Wozz". It was a most unusual country and a most unusual community. Country and community grew in importance as the imagination dwelt on them. This land was said to lie in the vastness of the Pacific Ocean, somewhere in what is known as the International Date Line. This has possibly had something to do with its being but little known. The traveller in one direction who loses a day would not be particularly concerned with a place called Wozz which would add to what he had already lost—he might tell himself that he had lived on a day that, for him, did not exist. The traveller in the other direction who gained a day might think that here was something to help him fill in his extra day, but the name would be likely to deter him. Wozz was said to be completely circular in shape, something like a gramophone record. In the centre was the capital city "Izz". The name must have been chosen by persons with philosophical insight who knew that at the centre of every "was" is an everlasting and unchanging "is". It was even said that the circular island seemed to revolve around "Izz"—that north, south, east and west, as known to the rest of the world, did not exist. Directions were calculated from the centre of Izz. Here lay the city square, on one side Science Centre and on the other the official residence of the *Princeps* or chief citizen, chosen in democratic fashion by an ideally democratic community. If one stood on the threshold of Science House and faced the palace of the *Princeps*, one spoke of four directions—right, left, facial and caudal. Of course, complications sometimes arose if the person so standing was left-handed or ambidextrous or had complete transposition of viscera. Still, by and large the "right, left, facial and caudal" worked very well. Of the size of this country or the number of its inhabitants nothing definite can be stated; no surveyors' activities have been reported and as far as is known no census was taken.

Of the activities of the philosophical and scientifically minded community, as it appears to be or to have been, we know most of the medical aspect and it is this that has prompted the present reference. The Wozz Medical Association claimed as members every medical practitioner in the community; they joined not by compulsion but of choice. The Association had two objects —first, the advancement of the medical and allied sciences, and secondly, the maintenance of the honour and interests of the medical profession. The two objectives were not confused; neither was any consideration put before the advancement of knowledge. In fact, scientific gatherings were always crowded. So keen were the practitioners to attend that a roster had to be drawn up setting out the names of practitioners who were to be on call for patients during scientific sessions. When questions of Association procedure or of fees arose it was quite common to find the meetings without a quorum. If no quorum was obtained at a second meeting called for a particular purpose, those present were deemed to constitute a quorum. Subjects for discussion at scientific meetings were carefully selected. As a rule persons of known attainment were asked for contributions; if a contribution was offered from an unknown member, it had to pass the scrutiny of a committee of selection before it could be presented at a meeting. Office-bearers of the Association were chosen with care. Presidents acted as chairmen only if they knew something about the rules of debate and if they were not given to the pernicious habit of taking part in debate from the chair. The community had its own medical journal, *The Medical Journal of Wozz*; it was not the official organ of the Association, but was controlled by a company elected by that body. The journal was known by its correct name; to call it *The Wozzian Medical Journal* was regarded as ill-informed and slovenly. A library with a competent librarian was to be found at Izz and members took a pride in the accuracy with which they used references from journals from other parts of the world. This was not a simple matter; it called for careful attention to detail as well as an appreciation of the value of a reference to anyone reading the paper containing it when it had been published. Being so far from other centres of medical activity the doctors of Wozz paid great attention to the making of complete arrangements for post-graduate study. A committee had charge of the arrangements and the practitioners themselves insisted that lectures and demonstrations should, if necessary, be repeated so that all might benefit from them. At all times the prevention of disease rather than its cure was kept in view. This was evidenced by the way in which the tuberculosis problem was tackled. The tuberculous patient was freed from the anxiety about what was to happen to his dependants while he was being treated. To do this was the easier because, as already mentioned, the community was a carefully planned democracy; people knew how to help themselves and to keep one another, and were prepared to do both. In such spheres of medical activity as obstetrics not only were maternity allowances paid, but help was provided in the home while the mother was in hospital and afterwards. Success was achieved here, as elsewhere, when the helps were not scientifically trained and were neither too young nor too attractive. Supervision for the growing child was provided by private and State practitioners and the supervision covered both bodily and mental require-

ments. In fact the advice of psychiatrists could be as readily obtained by any member of the community as advice on infections of, say, the respiratory or alimentary system.

The more important of the medical provisions for the people of Wozz have been mentioned. They may serve as an object lesson for other places. To produce worthwhile results, any medical provisions need to be appreciated and used in a proper and appreciative manner. The legendary character of Wozz in regard to its revolutions and fixed points of the compass reminds the medical worker that help in the conservation of a community may come from many quarters. The sun that rises today in one quarter may give promise of success; this success may or may not be obtained. Tomorrow a greater sun may appear in another quarter with even greater promise than the first. What is faced today with perhaps amazed enlightenment may be as nothing to what appears from behind at a later date. Whether these suns appear depends on the ability of the watcher to see or to sense them and it is in these abilities that the medical personnel of Wozz excelled. It is from Izz, the centre, that the changes were appreciable and it was there that influences could be most clearly felt. It is what is that counts. We need to lay hold on all that is good and to hold it fast. If our stronghold is in Izz we shall grow more like the practitioners of Wozz.

Current Comment.

ANOXIA OF THE NERVOUS SYSTEM.

SEVERAL aspects of anoxia affecting the nervous system have become prominent during recent years, particularly those related to aviation and to the sequels of *erythroblastosis fœtalis*. To these might be added the interest taken in carbon monoxide poisoning, which also has occasional sequels due to anoxia. Another variety of oxygen lack which has been brought prominently before the lay and professional public is that associated with congenital heart disease. This latter is the subject of an article by Joseph I. Mossberger, who reviews the literature briefly from the earliest periods up to the present, and then describes three cases which throw light on the anoxic lesions of the nervous system.[1]

It is evident on perusing quotations from writers ancient and modern how the dependence of life on breathing has held the imagination, both poetic and scientific, of many generations. Even Hector is recorded as suffering from what would now be called a "blackout". Aristotle was apparently the first to describe the difficulty in breathing at high altitudes. Unfortunately the errors of Galen early in the Christian era began a period of disordered thinking which lasted until the Renaissance, but from then onwards, till the appearance of Virchow with his cellular pathology, steady progress was made. The anoxia of high elevation was recognized as soon as balloon ascents were possible, and Mossberger quotes Tissandier's description of his own alterations in affective state followed by loss of consciousness at 26,000 feet. Modern work has added physiological teaching on the location and nature of the neural controls of respiration, and has provided chemical methods of analysis. The recent methods of cardio-vascular investigation by boldly catheterizing the heart have brought respiratory physiology to the ward and the operating theatre. As has often been the case in medical research, advances in knowledge have been made by the investigation of abnormal states. In the present instance useful information has been gained by examination of the nervous system in the living and after death in those who have suffered from severe hypoxia at high elevations, sequels of repeated applications of electric shock therapy, and the nervous trauma suffered by infants and young children as the result of *asphyxia neonatorum*, and congenital disease of the heart. Many writers have described constant changes in the cells of the brain following various degrees of hypoxia, and it is now well known that certain serious defects of the central nervous system are not due to developmental faults or to external damage, but to the ravages in these sensitive cells caused by lack of oxygen. Even the capacity of the cerebral cells to derive nutriment from glycolysis when supplies of oxygen are deficient cannot save all the cells affected if the deprivation lasts longer than a short time.

Mossberger describes in detail three children suffering from congenital heart disease who succumbed. One was newborn, one five months old, and one three years of age, the last-mentioned dying after an operation to relieve symptoms due to patent *ductus arteriosus*, with an aberrant subclavian artery on the right side. The details of the anatomical abnormalities of the hearts of these children need not be here pursued. The most interesting feature common to all is the presence of well-marked changes in the brain. Various parts of the central nervous system showed established changes revealed by Nissl staining. Neuronal degeneration and necrosis varying in severity and extent were found, and glial reactions also. Gross macroscopic changes were not seen. The author has compiled this review as a pendant to the account of these three children, who would have had degenerative changes in their nervous systems had they survived. He therefore stresses the importance of guarding the nervous systems of infants, born or unborn, against the malign effects of lack of oxygen. There is nothing new in this plea, but it comes at an opportune time, when anæmias of the newborn are of great interest, and when operations for the relief of congenital cardiac disease are almost daily events. When action of any kind is decided n in any of these conditions the effect of hypoxia on the nervous system calls for serious thought, for irreversible lesions can easily be produced. It may not be out of place to remark in conclusion that careful clinical survey of these little patients may give rewards in the form of useful information. The familiar posture of the young subject of congenital heart disease when he squats in response to relative over-exertion may be mentioned. How many must have observed this phenomenon without curiosity as to its physiological cause?

[1] *American Journal of Diseases of Children*, July, 1949.

VAGOTOMY AND PEPTIC ULCER.

VAGOTOMY as a measure in the management of peptic ulcer is being mentioned little in current medical literature, at least as compared with the prominence it was receiving about the time when reference to the work of Dragstedt was made in these columns on June 14, 1947. There are not wanting those who consider this operation a passing fashion which will be forgotten before very long. Whether they are right or not will depend upon the soundness of the theories on which the operation is based, the permanence of the encouraging results already reported, and the possibility of achieving the same results by simpler means. The theoretical basis of Dragstedt's work has been previously considered, and it is of interest now to call attention to investigations by R. Brummelkamp recently reported in a new Dutch journal.[1] Brummelkamp describes a series of experiments mostly carried out before World War II, partly in Indonesia, partly in Holland, and presumably not previously published because of the war. Briefly, he found that with rats, rabbits and cats it was possible to produce gastric ulcers in practically 100% of cases by ensuring that free gastric juice remained for a certain time in contact with the mucosa of the fasting stomach. If under otherwise identical experimental con-

[1] *Archivum Chirurgicum Neerlandicum*, Volume I, 1949, Number 2.

ditions the gastric acid was fixed, no ulceration occurred. The ulcers in the rabbit and the cat were comparable with those occurring in man as regards site, form and histological appearance. Brummelkamp refers to other factors often regarded as alternative to or coincident with autodigestion in the genesis of peptic ulceration, and points out the improbability that one or other of these could be reasonably expected to be present in each case in which an ulcer was produced experimentally. His experiments were relatively simple and straightforward, and while not conclusive they do support the view that in man also autodigestion and consequently ulcer formation in the stomach wall can occur if the conditions for prolonged contact of free gastric juice with the stomach wall are fulfilled. The effect of gastric juice secreted in response to stimulation associated with foodstuffs is cancelled out by the presence of the food, but cephalic (psychic) stimuli conveyed by the vagus can bring about a secretion of gastric juice in man during fasting and thus create the conditions found to be harmful in certain animals. Suppression of these stimuli would seem rational treatment of peptic ulceration, and this it appears can be effected by means of vagotomy. It is true, of course, as has been pointed out by George Crile, junior,[1] that the acidity of the gastric juice may be normal or even low in association with gastric ulcer, and Crile considers that, especially in view of the associated possibility of carcinoma, in most cases of gastric ulcer requiring operation gastric resection is to be preferred to vagotomy; vagotomy for gastric ulcer is not recommended unless the ulcer is excised and examined microscopically or the ulcer is so high that it cannot be resected without performance of total gastrectomy or the incurring of undue risk. The management of duodenal ulcer is, as Crile points out, a different matter; it is primarily a medical problem, and operation should not be advised until medical management has been given a fair trial. If surgical intervention is considered necessary, Crile, on the basis of two and a half years' experience, considers transabdominal vagotomy with gastroenterostomy or pyloroplasty the treatment of choice; he believes it to be safer than gastric resection, more effective in controlling the tendency to recurrent ulceration, and attended by less morbidity and disability. Crile has been associated with the performance of transabdominal vagotomy 228 times, only three deaths occurring which could reasonably be attributed to the operation. He quotes a personal communication from Dragstedt to the effect that he (Dragstedt) had performed approximately 300 transabdominal vagotomies with "no deaths attributable to the procedure". Crile agrees that his own period of observation has been too short to permit final evaluation of the method, but over a period of more than two years in his experience and five years in that of Dragstedt it "has afforded the best protection against recurrence of ulcer".

A different side of the picture is shown by T. Grier Miller[2] in a brief survey from the physician's viewpoint. It would not be fair to suggest that Crile, or indeed any other surgeon writing on the subject, has failed to refer to the disadvantages of vagotomy, and Crile is most cautious in defining its limited use for gastric ulcer, for which in general he prefers gastric resection. Miller, however, draws particular attention to the unsatisfactory results which are only too common in the literature. He admits that medical management of the refractory peptic ulcer is unsatisfactory. The most consistently good results have followed subtotal gastrectomy, being best when the patient had a gastric ulcer and worst when the patient was psychoneurotic and had a duodenal ulcer. The immediate results of vagotomy, Miller considers, are hardly so satisfactory as those of gastric resection. The relief of pain that usually results from vagotomy may in itself lead to a false sense of security, since in several instances it has masked a perforation. The depression of gastric motor function often accounts for a prolonged period of epigastric fullness and vomiting, even when gastro-enterostomy also has been performed. As yet no assurance can be given that the previous gastric acidity will not recur. The ultimate effect of the operation on the pancreatic secretion is still in doubt. Secondary or associated gastric surgery is frequently necessary and when a stomal ulcer once develops (as it may after gastroenterostomy) its management, even by gastrectomy, leads to poor results. At any rate, as Crile also has emphasized, vagotomy alone is usually contraindicated in gastric ulcer because of the difficulty in ruling out a malignant lesion. On the other hand, Miller goes on, vagotomy does seem to have found a place in the management of the marginal ulcer in which the other current operations often fail. Also it may be found useful for the psychoneurotic patient with a duodenal ulcer for whom gastrectomy now gives its poorest results; it may indeed here be a rational procedure, since the mechanism of ulcer production in such case probably depends primarily on a disturbance of the autonomic nervous system. Its place in the management of the ordinary duodenal ulcer, however, has not yet been determined and, until further observations have been made on the patients already submitted to vagotomy, it would seem wise to exercise great caution in subjecting to this operation more patients with this type of ulcer. Miller makes the deliberate suggestion that the time may have come to abandon temporarily the ready employment of vagotomy until a longer period of evaluation of results is available—a suggestion which seems to warrant the most serious consideration.

[1] *The Surgical Clinics of North America*, October, 1948.
[2] *The American Journal of Medicine*, August, 1949.

ACCIDENTS IN THE HOME.

THE prevention of accidents in the home is no new theme, but it never becomes out of date. It needs constant publicity—far more than it at present receives. The weight of accident-prevention activity in this country is largely thrown behind road safety measures, which are, of course, of great and pressing importance; but planned attention is needed also to accidents in the home—the accidents in particular of women and children and old people. In Britain the Royal Society for the Prevention of Accidents has done useful work in this field and has, amongst other activities, published a series of pamphlets on safety measures. In a recent article on the prevention of accidents in the home,[1] in which frequent reference is made to these pamphlets, it is pointed out that major factors involved in the high death and injury rates from accidents of this type are carelessness, the speed of the present-day mode of life and unawareness of the potential hazards in the home. The matter of unawareness is specially considered; many people assert that they "know" all the accident-prevention measures suggested to them, and this may be so, but if the knowledge has not been applied, its significance has presumably not registered, and the individual is in practice, if not in theory, still unaware of the hazard. After considering two important topics, the safety of babies and the prevention of fires, each the subject of one of the Society's special pamphlets, the author of the article takes us on a tour through the home—the place that spells security, the Englishman's castle—and we must be startled to realize that we are set in the midst of so many and so great dangers. The bathroom, bedroom, landing and passages, kitchen, sitting room and garden, each has its hazards, with corresponding safety precautions which we cannot try to detail here. Perhaps it would be of little value to do so, for, as is pointed out, the list could be expanded indefinitely and is best compiled for the individual home. However, two important things are to be remembered: firstly, that accidents are less likely to happen in well-ordered households where neatness and tidiness are the keynote, and secondly, that they are more likely to happen when the attention wanders, as it invariably will, or when full concentration is not being given to the task in hand. But constant education and advice are needed. There is probably no one better fitted and placed to provide this than the family doctor.

[1] *The Journal of the Royal Institute of Public Health and Hygiene*, July, 1949.

Abstracts from Medical Literature.

THERAPEUTICS.

Antithyreoid Activity of Para-Aminobenzoic Acid.

J. F. GOODWIN, H. MILLER AND E. J. WAYNE (*The Lancet*, December 31, 1949) state that the results of clinical tests, and the evidence obtained by the tracer technique in normal persons, seem to show that para-aminobenzoic acid has a definite antithyreoid action, probably sufficient to influence mild or moderate degrees of thyreotoxicosis, but significantly less than that of the thiouracil compounds. Patients with more severe grades of thyreotoxicosis are unlikely to be improved unless toxic doses are given. It is pointed out that methyl thiouracil has only one serious side-effect—agranulocytosis—whereas para-aminobenzoic acid has at least three potentially serious side effects—depression of the bone marrow, renal damage and fatty infiltration of the liver.

Therapeutic Criteria in Rheumatoid Arthritis.

O. STEINBROCKER, C. H. TRAEGER AND R. C. BATTERMAN (*The Journal of the American Medical Association*, June 25, 1949) present a report summarizing recommendations for uniform therapeutic criteria in rheumatoid arthritis; the criteria have been adopted by the New York Rheumatism Association and the American Rheumatism Association (Executive Committee). The authors state that the first consideration in undertaking the treatment of a patient with rheumatoid arthritis is to determine the state of the disease, the presence of rheumatoid activity and the degree of functional impairment. They put forward a definition of rheumatoid arthritis, and classifications of stages of rheumatoid activity and of functional impairment; these are supplementary aids to the detailed therapeutic criteria for response of rheumatoid activity, which it is recommended should be the only basis in reports on the evaluation of specific therapeutic agents.

Chloromycetin in Typhoid Fever.

A. T. COOK AND D. E. MARMION (*The Lancet*, November 26, 1949) report 25 cases of typhoid fever, in 14 of which (the most severe cases only) treatment with chloromycetin was given. The original intention was to carry out a controlled trial, but the first of the control patients became so desperately ill that it was deemed unjustifiable to withhold chloromycetin, and the authors consider that there is little doubt that it saved his life. Of the 14 patients treated with chloromycetin three suffered relapse, but responded to a second course. There were no deaths among those treated with the drug, but one occurred amongst those not treated. In general terms the effect of the chloromycetin was to terminate the fever within five days and to arrest the progress of the disease at the stage reached. No toxic effects were observed.

H. S. COLLINS AND M. FINLAND (*The New England Journal of Medicine*, October 13, 1949) report the effect of chloromycetin in four cases of typhoid fever. Clinical improvement and defervescence began on the third day after treatment had been initiated in each case and was complete after about a week of therapy. One patient had a relapse with bacteriæmia after the temperature had been normal for two weeks. Another patient continued to shed typhoid bacilli in the stools throughout three weeks of chloromycetin administration and again during convalescence. The drug, given in doses of two grammes a day for two weeks, failed to cure a chronic typhoid carrier whose gall-bladder had been previously removed.

Antihistamine Drugs and Convulsive Seizures.

J. A. CHURCHILL AND G. D. GAMMON (*The Journal of the American Medical Association*, September 3, 1949) state that two antihistamine drugs studied, "Benadryl" and "Pyribenzamine", are capable of inducing seizures in epileptic patients with focal lesions of the cerebral cortex. "Pyribenzamine" also increased *petit mal* seizures; "Benadryl" had the opposite effect, but no claim is at present made for its place in the treatment of *petit mal*. The authors point out that these agents must be used with care in treatment of patients with convulsive disorders, with the knowledge that they are capable of inducing seizures in such persons. They suggest that the effect may find use in activating or evoking the abnormality in electroencephalographic tests.

Streptomycin and Tuberculous Sinuses.

R. H. DAVIS (*The Lancet*, November 26, 1949) reports the giving of systemic streptomycin therapy to 11 patients with 26 draining cutaneous tuberculous sinuses, which in five cases originated in bone and in three followed thoracoplasty, in two nephrectomy and in one Monaldi drainage. He states that the treatment has a definite value for sinuses developing in thoracoplasty or abdominal wounds of tuberculous origin, and that the response in sinuses originating in bone is dramatic. He makes a plea for the treatment of tuberculous abscesses by early drainage under streptomycin cover, but states that further experience will be necessary to determine the best time for such drainage.

Influence of Heart Rate on Cardiac Output.

H. G. KELLY AND R. I. S. BAYLISS (*The Lancet*, December 10, 1949) state that when digoxin is given in cardiac failure the use in output is as pronounced in patients with sinus rhythm as in those with auricular fibrillation. There is no relation between the degree of slowing and the increase in cardiac output; relief of venous congestion also is independent of the degree of slowing. Improvement in cardiac output is equally probable whether the initial ventricular rate is slow or fast. In normal hearts atropine acceleration is accompanied by a fall in right auricular pressure and a rise in cardiac output. Patients with heart disease showed essentially similar responses; there was no evidence that acceleration so induced either depressed cardiac output or increased venous congestion. Given after digoxin in fibrillating hearts, atropine, it is stated, can reverse the slowing effect without counteracting either the rise in cardiac output or the fall in venous pressure caused by digoxin. Similarly in patients with sinus rhythm atropine acceleration has no adverse effect on cardiac output or venous pressure after digitalization. The presence or absence of cardiac slowing plays no measurable part in producing the immediate hæmodynamic improvements following digitalization.

"Sulphetrone" in Pulmonary Tuberculosis.

H. V. MORLOCK AND R. LIVINGSTONE (*The Lancet*, December 24, 1949) discuss the reports in the literature on treatment of pulmonary tuberculosis with "Sulphetrone". They describe the results of their treatment of 25 patients with the drug and, as a result, are of the opinion that there is no evidence that "Sulphetrone" *per se* has a place in the treatment of pulmonary tuberculosis.

Cutaneous Reactions to Insulin.

R. G. PALEY (*The Lancet*, December 31, 1949) states that the incidence of cutaneous reactions to insulin therapy in 147 patients attending a diabetic clinic was 55·8% and was not confined to any one brand of insulin. Most of the sensitive patients reacted to a test solution of commercial soluble insulin. The importance of minor accessory factors was demonstrated, such as the pH of the solution and the salmine sulphate. A strikingly significant difference was noted between the mean area of reaction to an intracutaneous injection of the patients' current batch of insulin and the smaller area from insulin recrystallized six times. It is postulated that some patients are sensitive to insulin protein itself. Alternatively, these patients may be sensitive to minute traces of secondary protein still lingering in the purified insulin. Treatment of distressing local insulin reactions with recrystallized insulin is recommended.

Hæmolytic Effects of "Myanesin".

D. R. T. CLENDON AND J. B. PENFOLD (*The Lancet*, November 26, 1949) review previous work on the possibility that "Myansin" may cause intravascular hæmolysis and hæmoglobinuria, and report results of examination of serum and post-operative specimens of urine of 20 anæsthetized patients receiving "Myanesin" and 10 not receiving "Myanesin". Hæmolysis was present in 18 of the test cases and in none of the controls. The differences in the two series were significant as regards casts and albuminuria and highly suggestive as regards hæmoglobinuria and red cells. It is suggested that in the present state of our knowledge of this drug it should be given in the smallest effective dosage.

Oxygen Effects in Chronic Cor Pulmonale.

C. E. DAVIES AND J. MACKINNON (*The Lancet*, November 12, 1949) state that oxygen, at pressures up to one atmosphere, is generally regarded as a safe and important therapeutic agent in congestive heart failure due to chronic disease of the lungs (chronic *cor pulmonale*). Recent experience, however, has suggested to them that oxygen may produce changes in the intracranial circulation of these

patients, and that untoward effects sometimes follow. They describe two cases in which this occurred; in one, oxygen was the probable cause of myoclonic arm movements, and in the other, it probably caused coma and may have contributed to death. Experiments showed that oxygen given in concentrations of 50% to 100% to patients with chronic *cor pulmonale* caused a sudden increase in cerebrospinal fluid pressure. Such an increase was not observed in controls.

Black Hairy Tongue and Penicillin.

S. A. WOLFSON (*The Journal of the American Medical Association*, August 13, 1949) reports four cases of black hairy tongue associated with penicillin therapy. Possible modes of action of penicillin in causing this condition are discussed.

NEUROLOGY.

The Late Results of Head Injuries.

G. F. ROWBOTHAM (*The Journal of Mental Science*, April, 1949) obtained a series of 1000 hospital records of head injuries and traced 430 of the patients concerned. Of these patients 122 (such as housewives and children) were not gainfully employed; the remaining 308 were selected for closer survey. Penetrating injuries were excluded. From the mass of material no statistical survey is attempted, but it is stated that persistent deteriorations in personality and in emotional stability are very common following head injuries. Employers frequently stated that men who had had head injuries were in need of constant supervision of their work. With few exceptions, it was stated by relations that money received in litigation in no way compensated for their suffering due to the change of personality. In the author's opinion such money should be spent in seeking good health and obtaining household amenities. He stresses the greater surgical interest necessary and the need for supervision of rehabilitation by the same person. The prognosis depends upon three factors: the nature of the injury, the type of man injured, and the nature of the subsequent occupation.

Forecasting the Incidence of Neurosis.

W. MAYER-GROSS *et alii* (*The Journal of Mental Science*, January, 1949) discuss a systematic method of estimating the propensity to neurosis of officer candidates or applicants for comparable executive or managerial positions. Information was collected from case histories of 100 army and 100 naval officers who had been treated for neurosis; 55 volunteers from a mobile army division were used as controls. From this material the evaluation of sixteen "pointers" was made. These were combined into three groups: (*a*) heredity, physical ill health, neurotic traits in childhood, unstable work record; (*b*) former psychiatric illness, instability, alcoholism; (*c*) adolescent shyness, adult shyness. Other factors noted included defective intelligence, sexual difficulties, head injury and war service. The three main groups form the basis of three short tests of tendency to neurosis. The first group can be considered indicative of inadequacy; the second indicates instability and has considerable diagnostic significance; the third is indicative of shyness (it has no independent value). The authors point out that any attempt to estimate the probability of neurosis by simply counting the pointers exhibited will be unsatisfactory. They have evolved specially adapted methods of statistical analysis to discover the weight and significance of the pointers, singly or in combination. They suggest that in this way psychiatric clinical material could be used in a similar manner to psychological test material.

Experimental Seizures.

C. L. ANDERSON (*The Journal of Nervous and Mental Disease*, March, 1949) points out that research into literature reveals 60 proposed causes of convulsive seizures, running the whole alphabetical gamut from acid-base balance, alkalosis, anoxæmia—through destruction of cerebral tissues, dehydration, hydration—all the way to vasoconstriction. He discusses the relationship of low blood cholesterol levels to epilepsy and the possibility of producing convulsive seizures by dissolving some of the cholesterols of the brain in a living animal. He states that acetone was injected into the cerebro-spinal fluid of rabbits without damage to the brain substance. Each injection was followed by a major convulsion. A tentative conclusion is drawn that the convulsion was due to the immediate fat solvent effect of the acetone.

Morale and Flying Experience.

D. STAFFORD-CLARK (*The Journal of Mental Science*, January, 1949) gives an account of the various forms of flying stress to which bomber command crews were subjected, and of the effects which these had on morale. Observations were made on over 4000 pilots between 1941 and 1946. Two aspects of morale were considered: the basic attitude of the men to their job and the changes which that attitude underwent in any particular individual during final flying training and operational tour. It was found that on any operational tour morale would be high just after the beginning, would tend to slump at any stage between the eighth and fourteenth mission (when full realization of the dangers involved was attained), would rise as confidence and skill increased and the end of the tour came into view, and might fall sharply just before the end of the 30 missions because of cumulative stress and resultant fatigue. All with psychogenic symptoms tended to fall into one of four groups, corresponding to the rise and fall in morale just mentioned. Group A broke down before commencing or just at the start of the tour; their attitude to flying and its risks was never related to reality and the prognosis was always bad. Group B broke down any time after the eighth mission; they exhibited characteristic anxiety reaction and were often extremely conscientious; the prognosis was usually good if they could be assisted over the crisis. Group C broke down near the end of the tour, owing to emotional fatigue; they drove themselves unmercifully and were often mildly obsessive; if they were observed in time and given adequate rest and change, the prognosis for further flying was good. In Group D the breakdown was precipitated by exceptional strain, sometimes the result of a single terrifying experience, and unless they improved greatly within three days from the development of the acute reaction, chances of completing the tour were small. Fear is the underlying factor in all the reactions, whatever the outward form. It is balanced by courage, confidence and resolution, which make up morale. Despite the intensity of stresses involved, they led to breakdown in only 5% of bomber command crews.

Early Reversible Paranoiac Reactions.

H. ROSEN AND H. E. KIENE (*The Journal of Nervous and Mental Disease*, April, 1949) have analysed 14,000 psychiatric casualties in the 96th (United States) General Hospital and discovered only eight cases of paranoia. Six are described in detail. The authors state that it is significant that five of the patients were nurses, one was a medical officer and two were line officers. The patients fell into a definite category. They were all rigid, perfectionistic individuals, whose psychoses were apparently precipitated by sudden promotions to positions of authority. All had suffered emotional insecurity early in life, showed unsatisfactory sexual adjustments, felt basically inferior, inadequate and insecure, and manifested a craving for recognition unattainable in the environment in which they found themselves, especially when the fact is taken into consideration that the military environment itself, under certain conditions, is even more rigid and unyielding than the patients' own rigid and unyielding personalities. It is surmised that the relative frequency in nurses was due to their receiving direct commissions in the army and being in a better position than most to continue their own paranoiac way without being removed from post to post. It is pointed out that individual psychotherapy is of little value. None the less the patients appear to adjust themselves fairly well when removed from the situation which sets the psychosis in motion. The authors briefly discuss prophylactic treatment. They are of the opinion that the elements of paranoiac behaviour should be studied at school, so that potential sufferers would have insight into the factors underlying their type of persecutory ideas.

Follow-Up of Electric Shock Treatment.

JAMES K. MORROW AND JAMES P. KING (*The American Journal of Psychiatry*, May, 1949) have studied by *questionnaire* the late results of 503 patients given electric shock treatment. It is suggested that in severe cases of the affective disorders treatment should be started as soon as the condition is diagnosed. By this procedure general health is maintained, exhaustion is prevented, long stay in hospital is avoided and the risk of suicide is diminished. In this last regard it is stated that there is an increased risk of suicide after the first one or two treatments. Though neither electric shock treatment nor insulin finally answers the therapeutic problem of schizophrenia, the effectiveness and ease of administration of convulsive therapy warrant its use as the first measure in schizophrenia.

Special Article.

BRISBANE AND SOME OF ITS HISTORY.

WHEN the Australasian Medical Congress (British Medical Association) is held in Brisbane in May, it will be early winter in Queensland. Brisbane's floral emblem, the poinsettia, will be in full flame. And if May be true, it will be a mellow month that should give warm fiction to the season's name.

It is said that Queenslanders are a friendly breed. I like to think they are, being one of them. Medical men may have a theory for it, if it be so. Climate, perhaps? The psychological effects of sunshine in all quarters of the year?

At any rate, Brisbane is a city of more leisurely living than the larger capitals of Australia. The pace of its comings and goings is slower. And wagers have been won — and lost — by people who say they can pick Queenslanders by their slightly different accent of speech, which, after all, should not be so surprising in a country of vast distances.

If Brisbane lacks some of the natural and acquired beauties in which some of the sister capitals are wrapped, it has, nevertheless, its own distinctive charm. If you were to view it from the most favourable lookout on Mount Coot-tha, you would see the river winding through an undulating vista that inspired George Essex Evans, the Queensland poet, to write of:

An amphitheatre of purple hills
And emerald slopes where nestling villas gleam,
Flooded with golden light that crowns and fills
Height, vale, and stream.

This was the river which Cook by-passed in 1770 and Flinders failed to find in 1799, and which Oxley came upon in 1823 only when two white castaways, Finnegan and Pamphlett, had directed him to its mouth. Such are the vagaries of history that a stream which did not obtrude into the earliest explorations has become Australia's largest commercial river.

FIGURE I.

The University of Queensland: looking through the cloister joining the Main Hall and the Chemistry Building.

By courtesy *The Telegraph*, Brisbane.

Much of the riverside richness was dissipated by pioneer failures to reserve the banks for park and drive. But the Brisbane City Council is going ahead now with schemes of beautification that find expression in really grand sweeps of river road, such as those at Coronation Drive and at Hamilton.

Within six years it is hoped to have trebled the 14,000 trees that now embroider many of the streets of suburban Brisbane—streets livened by bauhinia, hibiscus and oleander, and in late months of the year, splashed with the blue of jacarandas and the vermilion glow of the *Poinciana regia*.

Spanning the city reaches are three bridges—the Story Bridge (designed by the late Dr. J. J. C. Bradfield), Grey Street Bridge, and Victoria Bridge. Story Bridge, opened in 1940, is by far the largest, but the Grey Street Bridge is æsthetically the most pleasing. Eventually a fourth bridge will be built to connect the new university with the city at West End.

Visitors to the congress will probably see only the south-eastern section of the State around the metropolis. That is not Queensland. Here in the capital we are scarcely over the border from New South Wales. Though many citizens themselves do not realize it, in Brisbane they are slightly closer to Melbourne than they are to Cairns. And Cairns, in turn, is more than 450 miles from the tip of Cape York Peninsula.

But visitors will find in the capital, as everywhere in Queensland, a fervent, almost aggressive belief that the State is destined to be the richest and maybe the most populous in Australia when it comes to full productive maturity.

It certainly is favoured by Nature. Along the coast north from Mackay there is a rich tropical province of 100,000 square miles, the successful development of which under the white man's hand is held as practical justification for the maintenance of a wholly white population in Australia.

Queensland possesses almost half the pastoral lands of the Commonwealth that are classed as good because of rainfall. It is the great cattle State. Only now is it coming to realization of the immense coal deposits lying at shallow depth between the Callide Valley (inland from Rockhampton) and Bowen farther north. They are deposits capable of huge-scale working by open cut, and their prospective contribution to the industrial future of Australia is immeasurable.

Scarcely more than a modest start has been made in the storing for irrigational use of the water that bursts the banks of Queensland rivers in the monsoonal "wet" each year. The future does, indeed, hold high promise.

But if Queenslanders are State-conscious, they are none the less good Australians. Their fathers and grandfathers had a large part in forming the Federation. It was while returning from a heartening visit to the Premier of Queensland (Sir Thomas McIlwraith) in 1889 that Sir Henry Parkes, impatient of Victoria's delays in entertaining his Federal proposals, made the historic speech at Tenterfield that launched the campaign for creation of the Commonwealth. The first Federal Convention was held in Melbourne the following year.

Queenslanders like to recall that one of their own distinguished jurists, Sir Samuel Griffith, was among the chief architects of the Constitution, and lived to be its prime custodian as first Chief Justice of the High Court. Much of the Australian Constitution was drafted in his historic home, "Merthyr", in the Brisbane suburb of New Farm. At one time visitors to "Merthyr" could see a round table bearing the burn-mark from a cigar which one of the Federal framers had placed there forgetfully in one of their

scholarly sessions. Where is that table today? Old "Merthyr" itself has been turned into flats.

Be it confessed, many things of historic value have been lost to Queensland, for we are still without a State archivist, whose business it should be to collect old papers and records. But in the John Oxley Memorial Library and in a few other public and private collections there are some precious links with the past.

At North Quay there is a granite monument with a bronze plaque, announcing that: "Here John Oxley, landing to look for water, discovered the site of this city, 28th September, 1824."

We know, of course, that Brisbane became the capital by accident. As Surveyor-General of New South Wales, Oxley had been sent up by Governor Sir Thomas Brisbane to examine Moreton Bay for its possibilities as a new penal settlement to relieve the crowding at Port Macquarie and Botany Bay.

The site he originally selected in 1823 was at Redcliffe, now the largest of Brisbane's bayside resorts. The first batch of thirty convicts arrived there a few months later, but by the time Oxley had returned in 1824 to carry on his exploration of the river, aborigines and mosquitoes had so plagued the Redcliffe settlement that it was already in sight of abandonment. And so an up-river landing to replenish a ship's tank led to the founding of a metropolis.

In the years that followed, Cleveland, in the Redland Bay district south from Brisbane, was to be a rival for the primacy in settlement. And for a long time after Brisbane was founded, Ipswich, miles up the river, was the recognized commercial centre of the colony, with a busy boat trade to establish its claim.

When it came to the negotiations that led to separation from New South Wales in 1859, some influential colonials were advancing the cause of Port Curtis (Gladstone) and its magnificient harbour. But Queensland's first customs house was built in Brisbane, and that seems to have decided the issue.

There are people of tender conscience today who do not like to refer to the sad origins of Brisbane. I do not share their view. The seeds of this city, like those of most of the early Australian settlements, were sown in a bed of punitive misery, and there is no escaping the fact. Deplore it we may; cancel it we cannot.

There is, on the other hand, reason for pride in the fact that the penal place which became a colony for free settlers after 1840, and a municipality in 1859, is now a flourishing city of 405,000 people and the capital of a great and growing State of 1,110,000. You will agree that a million inhabitants is not many in a State of 670,500 square miles, especially when more than one-third of them are concentrated in one city. Queenslanders are conscious of that, and of the need for distributing more settlers to the north if we are to justify the holding of Australia within sight of millions of multiplying hungry Asians. That is why there is in North Queensland today a movement for the creation of a new northern State.

FIGURE II. Aerial view of Brisbane.

By courtesy *The Courier-Mail*, Brisbane.

Since 1925, when 20 suburban councils were absorbed, Brisbane's local government has been in the hands of a Greater Brisbane Council whose area covers 375 square miles. The Lord Mayor and 20 aldermen are elected by direct vote on an adult franchise.

If you should come to the city by air you will land at the historic Eagle Farm aerodrome. Historic, I say, because it was there in 1928 that Kingsford Smith and Ulm, touching down in the *Southern Cross* at the end of their ocean flight from America, broke through the dawn of a great day in pan-Pacific communications. At this same field, less than three months earlier, Bert Hinkler had arrived at the end of his remarkable solo flight from England in a frail aircraft

that now has a place in the Queensland Museum—a reminder of the enormous strides aviation has made since his triumphal day.

Speaking of aviation, it is worth recording, too, that Qantas (Queensland and Northern Territory Aerial Service) had its beginnings at Longreach (Queensland) in 1920.

Wickham Terrace, the medical quarter of the city, takes its name from Captain John Charles Wickham, R.N., first police magistrate and first Government Resident of the colony. His home, "Newstead House", is preserved under Government trust at Newstead as the historical centre of Queensland.

Brisbane's best-known landmark, the old Windmill, is there on Wickham Terrace, witness to the changes of 120 years. Convicts built it of sandstone blocks and hand-made bricks in 1829. The mill was meant to grind the early crops of grain from farms on the river flats below, but the wind sails were a failure. It was converted to a treadmill and was worked in that way for nearly eight years, usually by 25

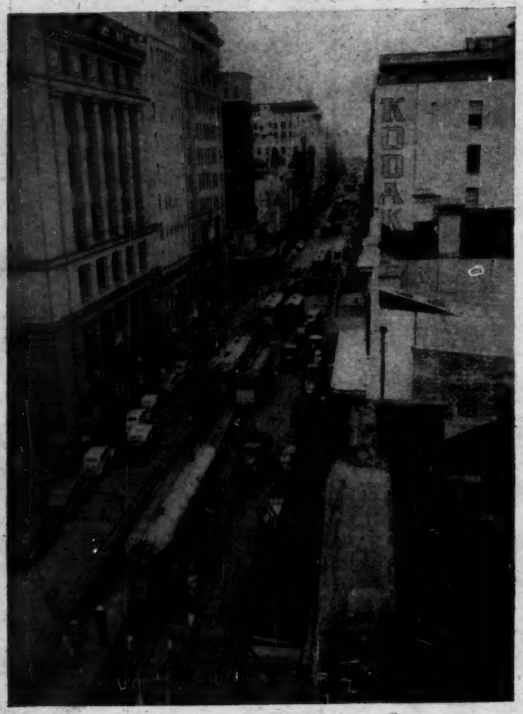

FIGURE III. Queen Street, Brisbane.
By courtesy *The Courier-Mail*, Brisbane.

prisoners at a time, but by fewer when they were undergoing special punishment.

Later the Windmill became an Observatory, and Brisbane used to set its watch by the raising of a bronze sphere on the flagpole and by the firing of a daily signal gun. In much more recent years the tower was used as an amateur radio station, where experiments in television and ultra-short-wave transmissions were carried out. Thus did the old become the servant of the new in a way the villagers of the 1830's could not have imagined.

When one talks of Brisbane's modern buildings, first place usually is given to the million-pound City Hall in King George Square. It is imposing, with its 300-foot tower and Corinthian columns, and its great circular concert hall.

Among the down-town buildings a special interest has been vested in the Australian Mutual Provident building at the corner of Queen Street and Edward Street, for it was from there that General Douglas MacArthur directed the South-West Pacific campaign against the Japanese from July, 1942. A tablet at the entrance to the building proclaims it for posterity. And inferentially it also marks the fact that Brisbane during the Pacific War was virtually a garrison town, through which passed 2,000,000 American troops, in addition to hundreds of thousands of members of the Australian forces.

Of more particular interest to the medical profession is the Brisbane Hospital. Embracing the General Hospital, the Children's Hospital and the Women's Hospital on 37 acres of ground, it is the largest institution of its kind in Australia. The matter of its size is merely recorded here, without any pretensions to discussing the advantages or disadvantages of hospital concentration. Suffice it to say that in the general section of the hospital alone the number of in-patients rose from 7200 (a daily average of 374 occupied beds) in 1924-1925 to 29,300 (a daily average of 1540 occupied beds) in 1948-1949. In the same time the number of new out-patients rose from 9900 (41,500 attendances) to 92,700 (336,700 attendances). The city's population in 1925 was 253,000, or roughly 60% of that in 1949.

In the Brisbane group of hospitals and the four small country hospitals controlled by the Brisbane Hospitals Board there are 75 full-time and 90 part-time medical officers. The Queensland Government dispensed with honorary medical service in its public hospitals several years ago.

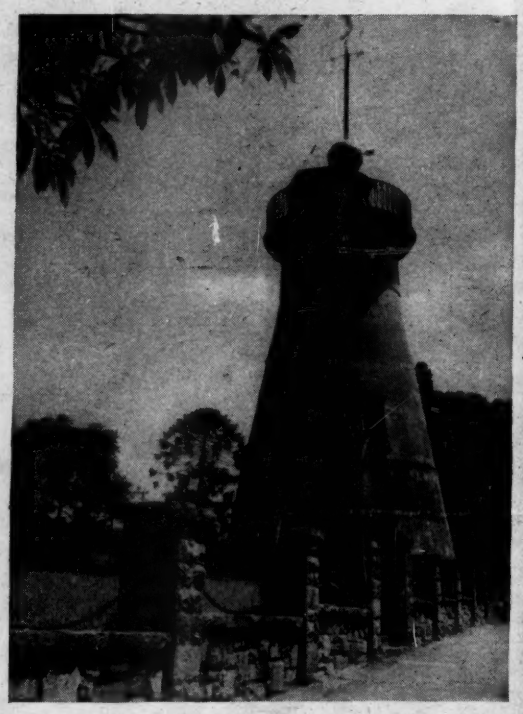

FIGURE IV. The old Windmill, Wickham Terrace.
By courtesy *The Courier-Mail*.

Close to the hospital, on Herston Road, is the University of Queensland Medical School. The building was opened in 1939, but the Faculty of Medicine had been established three years earlier, to the gratification of leaders of the medical profession who had long hoped and worked for it. The opening of the Medical School was one of the greatest advances in the university life of Queensland. Before 1936, students had had to go interstate or overseas to graduate in medicine. The Medical School building was planned to accommodate 105 students in the clinical years; in 1948 there were 166. Graduates in the Faculty of Medicine in 1948 numbered 271.

Reference to the Brisbane Hospital would be incomplete without a brief history of its earliest doctors, whose work made possible its foundation almost from the beginning of settlement in Moreton Bay. For the information on these men and their labours I am indebted to the researches of the late Dr. E. Sandford Jackson. This, in essence, is their story.

There arrived with John Oxley's party in 1823 a Mr. Cowper, who, it is now accepted, was the first medical man in the colony. He began his work among sick soldiers and convicts in a few tents which he himself had helped to pitch. He worked in the settlement at least until 1838, but at some stage before 1830 was given the assistance of a Dr.

Murray. The signatures of both of them appear on early medical records dispatched to Sydney. About 1838 the name of a Dr. Alexander, who was attached to a garrison regiment, is also mentioned.

Then, in March, 1838, came Dr. David Keith Ballow, a jovial Scot, to take charge of Her Majesty's General Hospital in Moreton Bay. The first hospital had been opened about 1825 on the site of the present Supreme Court of Queensland, between George Street and North Quay. It was there until 1866, when the first buildings on the present hospital site at Bowen Bridge were opened.

If you were to dig deep enough among the oldest records of the institution you would find the grim entry *"flagellatus"* in columns giving the cause of illness beside some of the convict patients' names.

Now, Dr. Ballow's appointment was primarily a military one; but he came in time to see the removal of convicts and the advances towards free settlement, so that he emerged also as a civil practitioner—the first, I should say, in what was to become the State of Queensland.

By 1843 Dr. Stephen Simpson and Dr. Kearsey Cannan had arrived to share his company and his work. Simpson had a colourful career. For a time he interspersed with his medical practice the duties of Commissioner for Crown Lands. In 1848 he was a trustee, along with Dr. Ballow, of the Brisbane Hospital. The hospital then was ceasing to be a military institution because of the pending withdrawal of the soldiery. In 1860 Dr. Simpson became a member of the first Legislative Council of Queensland. Nine years later he died in London.

At the first meeting of subscribers to the Brisbane Hospital after it became a public institution in 1849, Dr. Ballow became its first resident surgeon at £25 a year *plus* quarters—a salary soon raised to £40 a year—and Dr. Cannan was appointed visiting surgeon at £30 a year. They had the right of private practice.

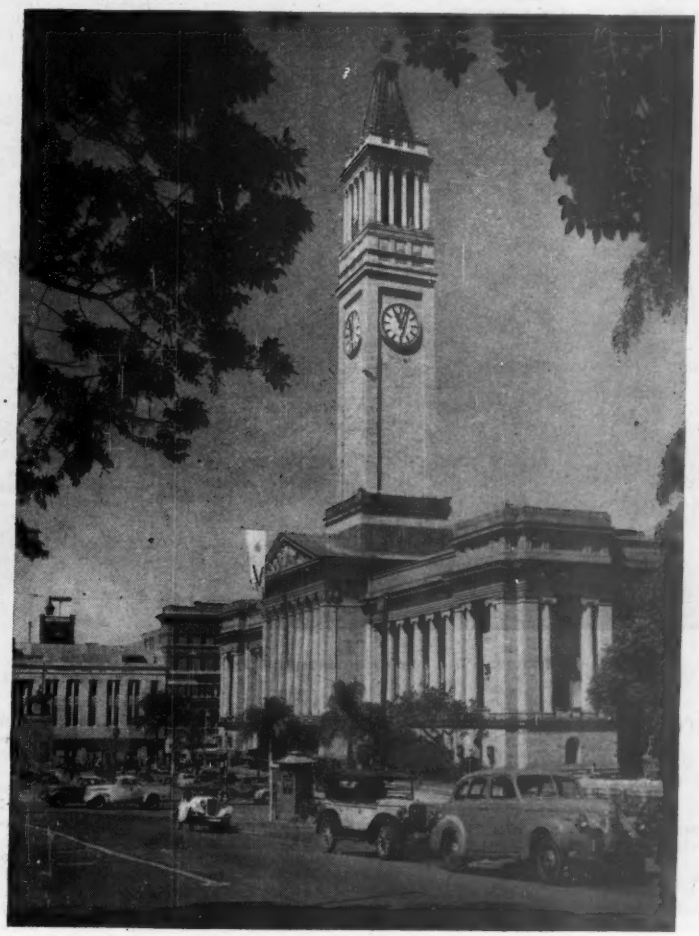

FIGURE V. City Hall, Brisbane, and King George Square.
By courtesy the Lord Mayor of Brisbane.

Ballow, Cannan, Simpson. They were the founders of the great hospital that serves Brisbane today. They were the banner-bearers of Queensland medicine who deserve special tribute at a time of such significance as the holding of the coming congress.

Dr. Ballow's memory should be held in particular reverence, for he was a man who died in his duty. In September, 1849, he went down to meet a ship in quarantine at Dunwich (Stradbroke Island). The ship's surgeon had been stricken with typhus, which raged among the passengers. The ship's surgeon recovered, but Ballow himself contracted typhus and died. At his death Dr. Cannan went down to Dunwich and took charge of the outbreak.

In that same year there came to Brisbane another medical man who was to figure prominently in the early history of the town. He was Dr. Hobbs, who arrived a surgeon in the immigrant ship *Chasely*. His home is now the Deanery of Saint John's Anglican Cathedral.

Last, but not in importance, among the notable institutions of Brisbane that merit mention in what must necessarily be a summary view, is the University of Queensland. When the "locals" speak of the university these days they think of the proud pile that is rising on 240 acres of land above the Brisbane River at Saint Lucia. But really the university is in a state of transition.

It is young. Its foundation dates from December, 1909, during the governorship of a distinguished medical man, Sir William MacGregor, who became its first Chancellor; but historians place its birth in 1910. Before he came to Queensland, Sir William MacGregor had been Administrator of the Territory of Papua, where he made notable explorations and did outstanding clinical and research work among the natives. Queensland was fortunate in having had such a man as first leader of its university. His name is perpetuated in the MacGregor School of Physiology.

The university's beginning was humble, notwithstanding that it was set in vice-regal surroundings. It was accommodated in the old Government House, Sir William and Lady MacGregor having gone up as first occupants of the present Government House on the heights of Paddington. As the university grew, the senate was obliged to use part of the adjacent buildings that had been erected for the Central Technical College. It added temporary buildings in the grounds. All this was makeshift.

Pleasant though was the setting on the fringe of the Botanic Gardens, old Government House was never intended to be a permanent home for the university, and the senate was relieved and heartened when the generous gift of the Saint Lucia land came to it from the late Dr. J. O'Neil Mayne and Miss Mayne, his sister, in 1926. They gave £60,000 for its acquisition.

Work on the new buildings was begun in March, 1938. The State Government undertook to provide £500,000 spread over five years for completion of the main buildings. The first three units were to have been ready for occupation in March, 1943. But the planners did not count on war. Work was interrupted in 1942, when the partly finished buildings were taken over as headquarters of the Commander of Allied Land Forces in the South-West Pacific (General Sir Thomas Blamey). Parts of the buildings have been used by the university since 1948, but Saint Lucia remains something for the future.

More than £750,000 has been spent on structure and grounds so far, and the work is going steadily ahead. The cost of the completed university, exclusive of the bridge to connect it more directly with the city, is expected now to be £2,000,000. The original estimate was £1,140,000. Independent of this, of course, will be the erection of six residential

colleges—five church colleges and the Women's College—which now live a cramped life in temporary quarters in scattered parts of the city. Sites for them have been allotted along a reach of river directly behind the Main Hall.

It will be a great day when the whole of this stately design is worked out in reality. Want of space—not to mention the absence of great benefactions such as other Australian universities have enjoyed—has been a limiting factor in the work of the University of Queensland from its inception. And yet it can be claimed with justice that the university has done much to energize the life and enrich the character of this northern State. But that which is forming in symmetries of freestone at Saint Lucia holds the promise of greater things. It is a symbol of that wider learning through which our student youth may work to the attainment of a truly great national culture in the years ahead. It is more than a symbol. It is a prophecy.

H. J. SUMMERS.

FIGURE VI. Brisbane Hospital Buildings.

By courtesy *The Courier-Mail*, Brisbane.

Medical Societies.

MELBOURNE PÆDIATRIC SOCIETY.

A MEETING of the Melbourne Pædiatric Society was held at the Children's Hospital, Carlton, on July 13, 1949.

Sudden Death of Babies.

DR. KEITH BOWDEN read a paper entitled "Sudden Death or Alleged Suffocation in Babies" (see page 65).

DR. REGINALD WEBSTER, in opening the discussion, expressed his thanks to Dr. Bowden for his paper and for the opportunity of examining some of his very convincing microscopic sections. He ventured the opinion that the majority, if not all, of those present supported Dr. Bowden in his contention that asphyxiation, with the mental anguish which such a finding involved for the infant's mother or other attendant, had in the past been much too frequently invoked as explanatory of the tragedy of the infant found dead in bed. Experience of the resistance offered by babies to the administration of an anæsthetic had led Dr. Webster to believe firmly that unless the infant was profoundly asthenic, he would respond to the instinct of self-preservation and wriggle himself free of entangling bed clothes or other obstruction to his airway. To Dr. Bowden's tenacity in what might be termed the "post-post-mortem" investigation of a series of infantile deaths which the routine autopsy had failed to elucidate, they were indebted for those illuminating findings which demonstrated poliomyelitis, miliary tuberculosis and diffuse interstitial nephritis in three babies respectively who had died without evident cause; the very interesting solutions of the problems must have amply compensated Dr. Bowden for the effort involved.

A situation which Dr. Webster had always found embarrassing and eventually learned to dread was that in which he had to devise, for the satisfaction of a group of medical students, some hypothesis or theory to account for the demise of a child apparently devoid of pathological change. That, he assumed, was the major problem of discussion, and he therefore proposed to omit from consideration such causes of sudden and unexpected death as congenital anomalies affecting the brain, the heart and the great vessels, or the several abdominal viscera, likewise internal hæmorrhage, intracranial, intrapulmonary and intraabdominal, which occurred in the neonatal period; excluded also were hydrocephalus, undiagnosed meningitis and clinically unobtrusive cerebral abscess or tumour, with all of which sudden death might be associated in older children.

Dr. Webster said that, rightly or wrongly, he had frequently expressed his opinion to the effect that the more dramatic the death, and the shorter the antecedent illness, the less the autopsy was likely to reveal. There could be no doubt that in many such catastrophes the lethal agent was a bacterium or virus, and that the infant had been overwhelmed before he could establish those reactive phenomena upon which the pathologist so much relied for morbid anatomical evidence. Concerning the basis for such failure of reactivity one could only speculate, but Kahn, in his book on tissue immunity, had adduced experimental evidence of a lack of immunity response in the very young; he found that if adult rabbits were injected subcutaneously with small doses of virulent streptococci or staphylococci, furuncles were formed, whereas young ten-day-old rabbits exhibited no local inflammatory lesions, but succumbed to a general septicæmia within twenty-four or forty-eight hours. Hence the importance of post-mortem blood culture, and the significance of relatively minor histological changes. Such should be sought for particularly in the trachea, bronchi, and pulmonary tissue, but Dr. Bowden's elucidation of unsuspected poliomyelitis and clinically silent nephritis indicated that the bounds of the histological field to be explored were very widely set, and the way of the pathologist was so hard that he was driven to wonder in what respect he had become a transgressor.

A statement which Dr. Webster could not base on his own limited clinical experience, but for which the authority was Hold's well-known text-book, was that in very young infants a fatal infection might run its course without producing noteworthy symptoms, even without fever. In such an event, if diagnosis was not established by blood culture, it might not be reached even when tissues obtained *post*

mortem were subjected to searching microscopic examination. Dr. Webster submitted that as a suggestion meriting consideration in cases of rapidly fatal and obscure illness in infants and young children, if blood culture had not been successful *ante mortem*, it should whenever possible be carried out immediately after death, before the body left the ward for the mortuary. The elimination of the usual eight or more hours' delay between death and autopsy would enhance the prospect of a positive finding and at the same time anticipate and probably dispel the reserve of those who considered that growth of organisms from the blood *post mortem* indicated nothing more than bacterial invasion of the tissues after death. It must be allowed that such objection to post-mortem blood culture was well founded when the organism recovered was one the normal habitat of which was the intestinal tract.

Reverting to the question of asphyxia, Dr. Webster emphasized that the post-mortem findings regularly associated with suffocation—the venous congestion, fluidity and dark coloration of the blood, and the subpleural petechiæ—were commonly observed when death had been preceded by convulsions, with the anoxæmia attendant upon them, and that apart from the inhalation of vomitus or mucus. Convulsions entered as a terminal event in a variety of infections, and might, of course, have a biochemical basis, such as hypoglycæmia or infantile tetany. It might be necessary therefore for the hard-pressed pathologist to seek the assistance of the biochemist; but it might legitimately be asked to what extent biochemical findings determined in the shadow of impending dissolution were vitiated by that circumstance. In a paper dealing with unexpected death in early life, Sydney Farber had reported having found on two occasions widespread hyperplasia of the islets of Langerhans, from which it appeared that hypoglycæmia was an important factor in the deaths of the children.

Dr. Webster said that very recently he had read a report relating to the sudden and unanticipated death of an infant who appeared to be doing well after bronchopneumonia. No gross cause for the sudden death was found, but examination of sections of the pancreas showed a most unusual but very decided localized hypertrophy of the islets of Langerhans in the head of the pancreas. Again the question arose of death from hypoglycæmia, but there had been no determinations of blood sugar by which it could be resolved. A review of the diverse conditions leading to hypoglycæmia was inappropriate at present, but it was interesting to note that in infants born of poorly regulated diabetic mothers, serious hypoglycæmia might develop within the first few hours after delivery, as the result of compensatory hyperplasia of the islet tissue of the offspring *in utero*.

Two conclusions to be drawn from Dr. Bowden's presentation were (i) that infants who died suddenly when apparently well, or affected with a presumed minor ailment, were often the subjects of serious unsuspected disease, and (ii) that the more the methods he applied were adopted and extended, the less would be the number of deaths referred to "pointing the bone", or to the *status thymicolymphaticus*.

FIGURE VII. University of Queensland Medical School.
By courtesy the Medical School (Mr. G. E. Thomson).

With reference to the thymic state, so long the haven of the harassed pathologist, it might be recalled that a special committee of the Medical Research Council and the Pathological Society of Great Britain and Ireland had concluded in a report issued twenty years previously that the facts elicited afforded no evidence that the so-called *status thymicolymphaticus* had any existence as a pathological entity.

DR. ROBERT SOUTHBY, after thanking Dr. Bowden for his informative paper, remarked that in most medical discussions the chapter was usually closed once the morbid anatomist and the pathologist had presented their findings. However, he hoped to present a small contribution to the discussion from the clinical aspect.

Dr. Southby said that sudden death might occur in two groups of infants: those who were apparently well and those who were ill but died unexpectedly suddenly. The family doctor was likely at any time to have to face the problem of trying to explain the sudden and unexpected death of a young infant. For example, a baby who had been born safely and appeared on the initial examination to be perfectly normal in every respect and whose parents had been assured that the baby was "quite well" died on the second day from a severe intracranial hæmorrhage. Another infant, aged five weeks, who had a blocked tear duct, was awaiting the arranged date to have the duct probed by an ophthalmic surgeon, but during that period the baby contracted a fulminating respiratory tract infection and died overnight. A third baby, aged twelve months, had celebrated her first birthday and appeared quite fit when seen the following day, but three days later had convulsions as she was having her midday meal and was dead by midnight the same evening from a fulminating meningococcal septicæmia. In each of the three households concerned the doctor was held to blame for not having warned the mother of the possible sudden death of her infant, although in every case the circumstances were entirely beyond his control or possible knowledge. It seemed an extraordinary trait in human nature that under such circumstances somebody must be blamed, and it was always the unfortunate doctor who received all the obloquy, the unfortunate parent adhering very firmly to her opinions.

Dr. Southby then mentioned a number of illnesses in which unexpected death might occur. Sudden death of athreptic or marasmic infants for whom a clinical diagnosis of failure to thrive or inanition had been made was not uncommon. Frequently those infants died suddenly just after they had been fed or changed, much to the concern of the nursing sister who had just handled the infant a few minutes previously. Such deaths might be associated with sudden myocardial failure, an acute infection to which the tiny infant had no resistance, or the very unstable nervous system of such infants. Convulsions might herald sudden death in infants as a result of overwhelming septicæmia, such as that of meningococcal or pneumococcal origin, or acute poisoning due to such substances as lead or phosphorus. In the frail baby suffering from *sclerema neonatorum*, death might occur quite unexpectedly, and the baby suffering from pink disease for some weeks might be found dead even when he had not appeared to be even seriously ill shortly before the *exitus lethalis*. Dr. Southby suggested that in pink disease the cause of death was most probably of a myocardial nature, since those infants frequently had a tachycardia with pulse rates of 180 to 200 per minute throughout the twenty-four hours for weeks at a time. Renal failure in the course of nephritis, or as a result of a congenital anomaly of the urinary tract, was another cause of unexpected death. The diabetic infant might succumb to sudden diabetic coma without the underlying cause having been suspected, or the baby having insulin could occasionally die in a hypoglycæmic attack. The infant who had had an unsuspected diphtheritic throat infection might suddenly collapse and die from myocarditis when he appeared to have completely recovered from the original sore throat. Congenital anomalies and hæmorrhage (either intracranial or due to hæmorrhagic disease of the newborn) would also explain some of the tragically sudden infantile deaths. The allergic infant was also a very unstable subject, particularly if he suffered from extensive eczematous dermatitis with superimposed infection or severe asthma with intercurrent respiratory tract infection. Those babies were particularly prone to develop hyperpyrexia under those circumstances with a rapidly fatal issue.

Dr. Southby said that Dr. Bowden's findings were almost identical with those recorded in England by W. J. Martin in a paper entitled "Mortality in Childhood during 1920-1938" (*British Medical Journal*, March 17, 1945, page 363) and by W. H. Davison in a paper entitled "Accidental Infant Suffocation" (*British Medical Journal*, August 25, 1945, page 251).

In concluding his remarks, Dr. Southby expressed the opinion that after Dr. Bowden's paper had been published in THE MEDICAL JOURNAL OF AUSTRALIA a short statement of his findings on sudden death in infants should be published in the lay Press. He considered that such a pronouncement to the public by Dr. Bowden, in his official capacity of Government Pathologist and Medical Officer to the Coroner, would contribute materially to the relief of the mental anguish of those distressed mothers who had erroneously been under the impression that they had been responsible for the death of their own infants by suffocation owing to carelessness or neglect. Such an impression must inevitably be indelibly fixed in the minds of those harassed women. It would further absolve the medical practitioner from the blame which was so often wrongly and unjustifiably placed upon him.

DR. V. L. COLLINS and DR. J. COLEBATCH both expressed the opinion that even though pathological changes were usually present in infants who had died suddenly, it was often difficult to explain the reason for sudden death and also the mode of sudden death, in the light of those morbid changes. Both speakers stated that Dr. Bowden had made a valuable contribution in so clearly demonstrating that sudden death of those babies was very uncommonly the result of accidental suffocation, and that there was usually definite evidence of unsuspected organic disease. By directing the attention of the medical profession to those facts much needless suffering could be avoided on the part of the mothers concerned, who often felt that they were on trial for neglect of their children.

DR. KATE CAMPBELL stated that she did not think that vomitus discovered in the bronchial tree of a baby who had suddenly died could be disregarded as being of little importance in causing death. She considered that in premature and weakly babies inhalation of food was often a major factor in causing death. In healthy babies she agreed that it was rarely of any importance as a cause of death.

Dr. Bowden, in reply, stated that he fully realized that his work was not complete. However, he considered that he had established three points: firstly that sudden death in infants was rarely due to suffocation, secondly that the circumstantial evidence of an infant found lying face down in the pillow dead was entirely unreliable evidence that accidental suffocation had been the cause of death, and thirdly that organic disease was commonly present in those infants. The better the methods used and the care taken in examining those babies, then the greater the number of cases in which the real cause would be discovered. He fully realized that the discovery of organic disease did not by any means always make clear how or why sudden death had occurred. With regard to vomitus in the respiratory tract in an infant unexpectedly found dead, he stated that that was such a common agonal event in patients dying suddenly from widely differing causes that, from the medico-legal viewpoint at least, he considered that undue attention should not be paid to the finding. He appreciated that clinically in a weakly, sickly baby or a premature baby, that might not be so.

Post-Graduate Work.

SCHOOL OF PUBLIC HEALTH AND TROPICAL MEDICINE (UNIVERSITY OF SYDNEY).

The Australian Diploma in Tropical Medicine and Hygiene.

THE next course for the diploma in tropical medicine and hygiene commences on February 13, 1950, and concludes with examinations commencing on or about July 10, 1950. This is a full-time course and gives extensive training in the subjects studied. The main subjects are tropical medicine, tropical hygiene, bacteriology and pathology, protozoology, helminthology, entomology and medical statistics. Practical demonstrations and instruction are also given in dermatology, ophthalmology and dentistry in aspects related to residence in the tropics.

The examination fee is £10. No fees are charged for tuition.

The course is recognized by the Colonial Office for appointment to its Colonial Medical Service.

Applications for enrolment should be made to the Director of the School of Public Health and Tropical Medicine (University of Sydney).

Diploma in Public Health.

The course of study for the diploma in public health is divided into two parts. Part I consists of essential basic training in such subjects as bacteriology, entomology and parasitology. Part II consists of the application of the knowledge gained in Part I to the control and prevention of disease and includes such subjects as hygiene, epidemiology, vital statistics, public health law and administration, sanitation and town planning.

An examination is held in each part and candidates must pass in the Part I examination before sitting for the examination in Part II. The course is full time, lasting three terms or one university year.

The fee for the diploma examination is £10. There are no tuition fees. Applications for enrolment should be made to the Director of the School of Public Health and Tropical Medicine (University of Sydney). The next course commences on March 14, 1950.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

LECTURES AND DEMONSTRATIONS IN ANÆSTHESIA.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that Dr. Ronald Jarman, of London, will be visiting Sydney in February. The following programme has been arranged.

Lectures to which all members of the medical profession are invited will be held as follows:

Wednesday, February 8, 1950: "Intravenous Anæsthesia", at the Stawell Memorial Hall, 145 Macquarie Street, Sydney, at 4.30 p.m.

Tuesday, February 14, 1950: "General Impressions of Anæsthesia as It Is in Great Britain Today." This second lecture has been arranged by the Section of Anæsthesia and will be held in the Robert H. Todd Assembly Hall, 135 Macquarie Street, Sydney, at 8.30 p.m.

Dr. Jarman will also conduct demonstrations in anæsthesia at the Royal Prince Alfred Hospital, Camperdown, on Monday and Tuesday, February 13 and 14, 1950, beginning at about 2 p.m. Attendance at these two demonstrations will be limited to anæsthetists only.

POST-GRADUATE COMMITTEE IN MEDICINE OF THE UNIVERSITY OF ADELAIDE.

PROGRAMME FOR 1950.

Annual Courses.

Refresher Week.

The refresher week will be held from August 14 to 18, 1950, inclusive. The programme will be advertised later. The fee will be £3 3s.

Week-End Courses.

A week-end course on the subject of peptic ulcer will be held on March 25 and 26, 1950.

A week-end course on pædiatrics and dermatology will be held to coincide with the visit of the American dermatologist, Professor A. Grace, who is expected to visit Adelaide in early September.

The fee for week-end courses will be £3 3s. During 1950 staff of hospitals and departments of anatomy, physiology and pathology will be admitted to all week-end courses for half fee.

Ward Rounds.

The weekly medical and surgical ward rounds will be given concurrently every Wednesday at 4.30 p.m. The fee will be £3 3s. per term of twelve sessions.

Histopathology and Post-Mortem Demonstrations.

Histopathology and post-mortem demonstrations will be given every Wednesday at 3.30 p.m. The fee will be £3 3s. per term of twelve sessions.

Terms.

Terms for these demonstrations and for ward rounds are as follows: first term, February 22 to May 10; second term, May 17 to August 2; third term, August 9 to October 25; (all dates are inclusive).

Higher Degree Courses.

Course for M.R.A.C.P.

A three-months full-time course suitable for candidates for the M.R.A.C.P. will begin on July 3, 1950. It will consist of clinical work, lectures, demonstrations, tutorials and "vivas". Further details may be obtained on application to the Medical Secretary. The fee will be 30 guineas.

Course for F.R.C.S. Part I or F.R.A.C.S. Part I.

A three-months part-time course suitable for candidates for Part I of the F.R.C.S. or F.R.A.C.S. will begin on July 10, 1950. It will consist of lectures in anatomy, physiology and principles of pathology based on the syllabus of subject matter required for this examination. The fee will be 30 guineas.

Both these courses will be held provided there are sufficient applicants. Graduates sitting for these examinations who do not wish to take the courses are advised to communicate with the Medical Secretary.

Special Short Courses.

If there are sufficient applicants short courses on special subjects may be arranged.

Overseas and Visiting Lecturers.

Edward Stirling Lectures.

Dr. F. B. Byrom, M.D., M.R.C.P., of Sydney, will deliver the Edward Stirling lectures on August 15 and 17, 1950. His subjects will be announced later.

Professor Arthur Grace.

Professor Arthur Grace, M.D., Professor of Clinical Dermatology and Syphilology, Long Island College of Medicine, New York, will give two lectures, one on dermatology and one on syphilis, during the first three weeks of September.

Overseas Lecturer.

The Australian Post-Graduate Federation in Medicine is making arrangements for the visit of the overseas lecturer for 1950. Further details will be published later.

Annual Ticket for 1950.

Admission to all the above lectures will be by annual ticket, which can be obtained from the Post-Graduate Committee Office, Institute of Medical and Veterinary Science, Adelaide.

THE MELBOURNE PERMANENT POST-GRADUATE COMMITTEE.

FULL-TIME COURSE IN PSYCHIATRY.

THE Melbourne Permanent Post-Graduate Committee would be glad to hear as soon as possible from those interested in a full-time course in psychiatry, which will be held in Melbourne in August, 1950, provided there are sufficient enrolments. It will be suitable for those studying for the diploma of psychological medicine and for psychiatrists generally, and will be the only course of its kind in Australia in 1950. It will consist of a concentrated series of lectures and demonstrations spread over one month and will be arranged by the Post-Graduate Committee in consultation with the Australasian Association of Psychiatrists.

Early inquiries from possible candidates will be helpful in the planning of the course. They should be addressed to the Secretary, Melbourne Permanent Post-Graduate Committee, 426 Albert Street, East Melbourne; telephone: JM 1547.

A TRAVELLING SCHOLARSHIP IN ANÆSTHESIA.

THE Association of Anæsthetists of Great Britain and Ireland has offered to the Australian Society of Anæsthetists a scholarship to enable an Australian anæsthetist to receive a year's training in Britain.

This gesture symbolizes the friendly relationship which exists between anæsthetists in the Mother Country and here. The scholarship offers a magnificent opportunity to the recipient. A graduate trained in the British way will obtain clinical and administrative experience which will be of benefit to the whole specialty of anæsthesia in Australia.

Applications will be called for this scholarship as soon as the conditions governing it are determined.

Correspondence.

SOME PROBLEMS ASSOCIATED WITH THE MANAGEMENT OF CARCINOMA OF THE BREAST.

SIR: Before this correspondence is closed, I would try to correct the harmful impression made by Dr. Kinsella's letter. I made the plea that the treatment of all cases of breast cancer should be planned by surgeon and radiotherapist in consultation, or at least that cases should all be sent for post-operative therapy. My one concern was that we should cure as many as possible of these women, who so often are in the prime of life. In particular, I am concerned that the many general surgeons who do the occasional mastectomy should be informed of the status of modern radiotherapy. I hope they have read Dr. Harold Ham's excellent letter and his contradiction of the wild statement that "the present evidence suggests that routine prophylactic . . . radiotherapy . . . is an unjustifiable gesture" *et cetera*. In actual fact, a correct interpretation of all the evidence has created in the minds of a world-wide majority of surgeons the impression that therapy is very important.

A closer examination of the reports of American workers quoted by Dr. Kinsella suggests they are not very reliable. For instance, the techniques used by Haagensen and Stout could not be expected to kill the cells of most breast cancers. Other American workers could be quoted who clearly demonstrate the effectiveness of radiotherapy in the cure of this condition, but Dr. Ham has referred to the radiotherapeutic work of McWhirter, of Edinburgh. This work appears to be beyond criticism, yet his results have been unequalled by any other method of treating the disease.

Dr. McWhirter has stressed some of the principles of modern therapy about which Dr. Kinsella inquired. They include the use of a more penetrating beam to avoid the excessive absorption in bones and ribs which occurs with softer X rays, and to attain homogeneity of dose over the whole field; the use of physical measurement and dose planning; fractionation over four or five weeks; the use of a routine dose near to the tolerance of normal tissues and skin. Unless therapy is of these standards, it only offers false security to the surgeon and cannot reproduce McWhirter's results.

A cancericidal dose, although important, is rather an arbitrary concept, because individual breast cancers show a considerable variation in their radiosensitivity. A routine dose, as high as possible, is chosen, lethal to the great majority of breast cancers, yet not causing permanent harm to tissues. A very small number of cancers are relatively radioresistant to this dose, and one accepts a small number of failures as a limitation of the method. I, too, have seen nodules appear on an irradiated chest wall, but much less often than when irradiation has not been given. The latter fact is a strong reason why therapy should always be given.

The optimum dose appears to be about two-thirds of the dose usually found effective in sterilizing squamous carcinoma. Because of the greater tolerance of a smaller area, one often exceeds this dose in treating an isolated metastasis, as Dr. Kinsella has noted.

It is quite true that accuracy is essential in prophylactic work, and it is quite attainable, with consistency. In practice, the radiotherapist attempts to do what Dr. Kinsella has regarded as impossible, that is, to give the desirable dose to each unit of tissue in the chest wall, internal mammary glands, skin flaps, axilla, retro- and supr[illegible]. This wide area just tolerates the optimu[illegible] dose is uniform it does not matter wh[illegible] actually lies. No man can yet dogmatize on [illegible] of radiation in affected internal mammary glan[illegible] the last few years are satisfactory techniques be[illegible] There is no doubt that the spread to these glands [illegible] the flaw in the otherwise sound operation of radical mastectomy, and is another good reason for using post-operative therapy.

Pre-operative therapy, either as a full course in more advanced cases, with delayed surgery, or else as a short course of low dosage in special cases immediately preceding the operation, is a measure found to be of value in those particular cases where consultation determines its use. Once again I advocate a radio-surgical approach to this disease, rather than just a surgical approach.

The time has passed when we should argue over the relative merits of radiation and surgery. Let us seek to collaborate to obtain the best treatment for each case.

Yours, etc.,

Perth, January 3, 1950. ALAN NELSON.

OCCULT BLOOD FROM CARCINOMA.

SIR: Can you or any of your readers tell me what percentage of tests show occult blood in the stools in the various carcinomata of the alimentary canal?

Yours, etc.,

118 Canterbury Road, Middle Park, Victoria. Undated. A. McQ. THOMSON.

"PREPARATION FOR MARRIAGE."

SIR: The Sydney Marriage Guidance Council has arranged for the Reverend W. E. Sargent, M.A., B.D., Ph.D., a psychotherapist consultant to the Marriage Guidance Council in Leeds, to speak on "Preparation for Marriage" at Bible House, 95 Bathurst Street, Sydney, at 8 p.m. on Monday, February 6, 1950.

Will you please let it be known that members of the profession are invited?

Yours, etc.,

143 Macquarie Street, Sydney, January 6, 1950. S. DEVENISH MEARES.

Australian Medical Board Proceedings.

QUEENSLAND.

THE undermentioned have been registered, pursuant to the provisions of *The Medical Acts, 1939 to 1948*, of Queensland, as duly qualified medical practitioners:

Giudice, Adamo Daniel, M.B., B.S., 1949 (Univ. Queensland), 86 Leichhardt Street, Brisbane.

Grant, Alan Gordon, M.B., B.S., 1949 (Univ. Queensland), Tintagel, King Arthur Terrace, Tennyson, Brisbane.

Gregory, Lee Westbrook, M.B., B.S., 1949 (Univ. Queensland), 61 Wirra Wirra Street, Toowoomba.

Harrison, Mark Robin, M.B., B.S. (Univ. Queensland), 2 Margaret Street, Toowoomba.

Hawkins, Warren, M.B., B.S., 1949 (Univ. Queensland), Pole Avenue, Northgate, Brisbane.

Hickey, William John, M.B., B.S., 1949 (Univ. Queensland), 24 Montpelier Street, Clayfield.

Howes, James Fraser, M.B., B.S., 1949 (Univ. Queensland), Flat 3, Kirston, Rupert Street, Windsor, Brisbane.

Isles, James Llewellyn, M.B., B.S., 1949 (Univ. Queensland), McLeod Street, Herston, Brisbane.

Jones, David Oliver, M.B., B.S., 1949 (Univ. Queensland), 167 Simpson's Road, Bardon, W.4, Brisbane.

Lahz, John Lister Colless, M.B., B.S., 1949 (Univ. Queensland), Wellwyn Crescent, Coorparoo, Brisbane.

Lennon, Evan Austin, M.B., B.S., 1949 (Univ. Queensland), 26 Kitchener Road, Ascot, Brisbane.

Loscher, Anthony Otto, M.B., B.S., 1949 (Univ. Queensland), 41 Virginia Avenue, Hawthorne, Brisbane.

McCabe, Patrick Glen, M.B., B.S., 1949 (Univ. Queensland), Carlton Terrace, Manly.

McGuinness, Edward Francis, M.B., B.S., 1949 (Univ. Queensland), 3 Henry Street, Ascot, Brisbane.

[illegible]eilson, Grahame Harrison, M.B., B.S., 1949 (Univ. Queensland), Rawnsley Street, South Brisbane.

O'Shea, Robert Francis, M.B., B.S., 1949 (Univ. Queensland), Ronville, Harriett Street, Kelvin Grove, Brisbane.

Perina, Allan George, M.B., B.S., 1949 (Univ. Queensland), Elmes Road, Rocklea, Brisbane.
Power, John Joseph, M.B., B.S., 1949 (Univ. Queensland), Whyenbah Road, Hamilton, N.E.2, Brisbane.
Ricketts, Graham, M.B., B.S., 1949 (Univ. Queensland), 87 Oxlade Drive, New Farm, Brisbane.
Shaw, Keith William Kirkland, M.B., B.S., 1949 (Univ. Queensland), Buderim, via Woombye.
Simpson, Roy Stewart John, M.B., B.S., 1949 (Univ. Queensland), 359 Margaret Street, Toowoomba.
Spiro, Harold Paul, M.B., B.S., 1949 (Univ. Queensland), Chez-Nous, Maxwell Street, New Farm, Brisbane.
Tait, William Howard, M.B., B.S., 1949 (Univ. Queensland), 45 Alfred Street, Mackay.
Taylor, Alan Graeme, M.B., B.S., 1949 (Univ. Queensland), 23 Pioneer Street, Toowong, Brisbane.
Weaver, Alexander Sydney, M.B., B.S., 1949 (Univ. Queensland), 440 Sandgate Road, Clayfield, Brisbane.
Weedon, Adine Pamela Jocelyn, M.B., B.S., 1949 (Univ. Queensland), 651 Sandgate Road, Clayfield, Brisbane.
Wilson, Graeme Brownlie, M.B., B.S., 1949 (Univ. Queensland), Main Avenue, Balmoral, Brisbane.
Zaver, Allan Gregory, M.B., B.S., 1949 (Univ. Queensland), 9 Rusden Street, Kelvin Grove, Brisbane.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*. Number 1, of January 5, 1950.

CITIZEN MILITARY FORCES.

Eastern Command: Second Military District.

Royal Australian Army Medical Corps (Medical).—2/50436 Captain J. J. G. McGirr is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (2nd Military District), 15th August, 1949. To be Captains (provisionally), 13th October, 1949: 2/127013 Edward Gordon Haig Manchester and 2/127012 Terence William Horne.

Southern Command: Third Military District.

Royal Australian Army Medical Corps (Medical).—3/51004 Major H. V. Francis, M.B.E., is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical) (3rd Military District), 16th July, 1949. To be Captain (provisionally), 13th October, 1949: 3/101010 Stanley Henry Watson.

Southern Command: Fourth Military District.

Royal Australian Army Medical Corps (Medical).—4/31956 Captain L. C. Lum is appointed from the Reserve of Officers, 9th September, 1949. To be Temporary Majors, 5th October, 1949: Captains 4/31905 C. M. Gurner and 4/31904 P. S. Eyles.

RESERVE CITIZEN MILITARY FORCES.

Royal Australian Medical Corps.

3rd Military District: To be Honorary Captain, 22nd September, 1949.—Bryan Harle Gandevia.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Rosengarten, Lionel, provisional registration, 1949 (Univ. Sydney), Flat 10, 69 Birriga Road, Bellevue Hill.
Claffey, Thomas Joseph, M.B., B.S., 1948 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.
Alchin, George Charles, provisional registration, 1949 (Univ. Sydney), 2 Knowlman Avenue, Pymble.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED DECEMBER 24, 1949.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.[2]	Australian Capital Territory.	Australia.[3]
Ankylostomiasis	*	3(3)	..	..	2	..	..	..	5
Anthrax	*	..	..	..	..	..	..	..	..
Beriberi	*	*	*	*	*	*	..	*	..
Bilharziasis	*	..	..	..	..	..	..	..	..
Cerebro-spinal Meningitis	3(3)	1(1)	..	1	..	..	..	..	5
Cholera	..	..	..	..	..	..	..	..	..
Coastal Fever(a)	*	*	..	*	*	*	*	*	..
Dengue	*	*	*	*	*	*	..	*	..
Diarrhœa (Infantile)	..	..	2(2)	..	..	..	..	..	2
Diphtheria	7(5)	3(2)	3(2)	..	3(1)	..	..	..	16
Dysentery (Amœbic)	*	..	..	..	..	..	..	..	..
Dysentery (Bacillary)	*	..	4(4)	..	3	..	..	1	8
Encephalitis Lethargica	..	..	..	..	..	..	..	..	..
Erysipelas	*	*	*	2	*	*	..	..	2
Filariasis	*	..	..	..	..	..	..	..	..
Helminthiasis	*	..	*	*	*	*	*	*	..
Hydatid	*	..	*	*	..	*	*	..	..
Influenza	*	*	*	..	*	*	..	*	..
Lead Poisoning	*	*	..	..	*	*	*	*	..
Leprosy	..	..	..	..	..	..	..	..	..
Malaria(b)	*	..	1(1)	..	..	..	..	..	1
Measles	*	*	*	38	*	*	..	..	38
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	18(2)	7(2)	..	31	2(1)	2	..	..	60
Pattacosis	*	..	*	..	*	*	*	*	..
Puerperal Fever	1	..	..	1	..	..	..	..	2
Rubella(c)	*	*	1	*	3(1)	*	..	..	4
Scarlet Fever	35(23)	7(3)	2(1)	7	5(5)	..	..	..	56
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	*	..	..	*	1	*	*	*	1
Trachoma	*	..	*	*	*	*	*	*	..
Tuberculosis(d)	14(12)	10(5)	4(2)	3	15(6)	7(2)	..	..	53
Typhoid Fever(e)	..	..	1(1)	..	..	1(1)	..	..	2
Typhus (Endemic)(f)	..	..	1	..	1(1)	..	..	..	2
Undulant Fever	..	..	..	..	..	..	..	..	..
Weil's Disease(g)	*	*	..	*	*	*	*	*	..
Whooping Cough	*	*	*	17	*	*	..	..	17
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] The form of this table is taken from the *Official Year Book of the Commonwealth of Australia*, Number 37, 1946–1947. Figures in parentheses are those for the metropolitan area.
[2] Figures not available.
[3] Figures incomplete owing to absence of returns from the Northern Territory.
* Not notifiable.
(a) Includes Mossman and Sarina fevers. (b) Mainly relapses among servicemen infected overseas. (c) Notifiable disease in Queensland in females aged over fourteen years. (d) Includes all forms. (e) Includes enteric fever, paratyphoid fevers and other Salmonella infections. (f) Includes scrub, murine and tick typhus. (g) Includes leptospiroses, Weil's and para-Weil's disease.

McDonald, George Roy William, provisional registration, 1949 (Univ. Sydney), 93 Victoria Road, Bellevue Hill.

The undermentioned have been elected as members of the New South Wales Branch of the British Medical Association:

Claffey, Thomas Joseph, M.B., B.S., 1948 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.

Fogl, Henry Walter, provisional registration, 1949 (Univ. Sydney), 206D Victoria Road, Bellevue Hill.

Hales, Ian Barnewall, provisional registration, 1949 (Univ. Sydney), 2 Jersey Road, Artarmon.

Hay, Judith Alison Ruth, provisional registration, 1949 (Univ. Sydney), Parramatta District Hospital, Parramatta.

Hunter, Irvine John, provisional registration, 1949 (Univ. Sydney), 7 Woodside Avenue, Lindfield.

The undermentioned have applied for election as members of the Tasmanian Branch of the British Medical Association:

Elrick, William Lindsay, M.B., B.S., 1949 (Univ. Melbourne), Royal Hobart Hospital, Hobart.

Telfer, Trevor Percival, M.B., B.S., 1948 (Univ. Melbourne), Royal Hobart Hospital, Hobart.

Kerridge, Gordon, M.B., B.S., 1943 (Univ. Sydney), Royal Hobart Hospital, Hobart.

Obituary.

WINIFRED ALICE McCLOY.

WE regret to announce the death of Dr. Winifred Alice McCloy, which occurred on December 18, 1949, at Strathfield, New South Wales.

EDWARD GREGORY BANNON.

WE regret to announce the death of Dr. Edward Gregory Bannon, which occurred on December 24, 1949, at North Balwyn, Victoria.

CHARLES WILLIAM ADEY.

WE regret to announce the death of Dr. Charles William Adey, which occurred on January 3, 1950, at Melbourne.

ROBERT ERIC HENRY.

WE regret to announce the death of Dr. Robert Eric Henry, which occurred on January 10, 1950, at London.

Corrigendum.

IN "Current Comment" in the issue of January 7, 1950, page 21, mention was made of a paper on heredity and retinoblastoma by Algernon B. Reese, but no reference was given. This paper appeared in *Archives of Ophthalmology*, August, 1949.

Medical Appointments.

Dr. John Catarinich has been reappointed Director of Mental Hygiene, in pursuance of the provisions of Section 5 of the *Mental Hygiene Act*, 1928, of Victoria.

Dr. R. M. C. G. Beard has been appointed gynæcological and orthopædic registrar at the Royal Adelaide Hospital, Adelaide.

Dr. C. R. Lulham has been appointed medical officer, Health and Medical Branch, Department of Health and Home Affairs, in pursuance of the provisions of *The Health Acts*, 1937 to 1948, of Queensland

Dr. H. A. McCoy has been appointed honorary radiologist at the Royal Adelaide Hospital, Adelaide.

Dr. N. C. Talbot has been appointed visiting medical officer, "Eventide", Rockhampton, in pursuance of the provisions of *The Charitable Institutions Management Act of* 1885, of Queensland.

Dr. J. M. McPhie has been appointed honorary clinical assistant to the officer in charge of the electrocardiograph at the Royal Adelaide Hospital, Adelaide.

Dr. J. S. Verco has been appointed honorary consulting radiologist at the Royal Adelaide Hospital, Adelaide.

Diary for the Month.

JAN. 25.—Victorian Branch, B.M.A.: Council Meeting.
JAN. 28.—Queensland Branch, B.M.A.: Council Meeting.
FEB. 1.—Western Australian Branch, B.M.A.: Council Meeting.
FEB. 1.—Victorian Branch, B.M.A.: Branch Meeting.
FEB. 2.—South Australian Branch, B.M.A.: Council Meeting.
FEB. 3.—Queensland Branch, B.M.A.: Branch Meeting.
FEB. 7.—New South Wales Branch, B.M.A.: Organization and Science Committee.
FEB. 10.—Queensland Branch, B.M.A.: Council Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135 Macquarie Street, Sydney): Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rate is £3 *per annum* within Australia and the British Commonwealth of Nations, and £4 10s. *per annum* within America and foreign countries, payable in advance